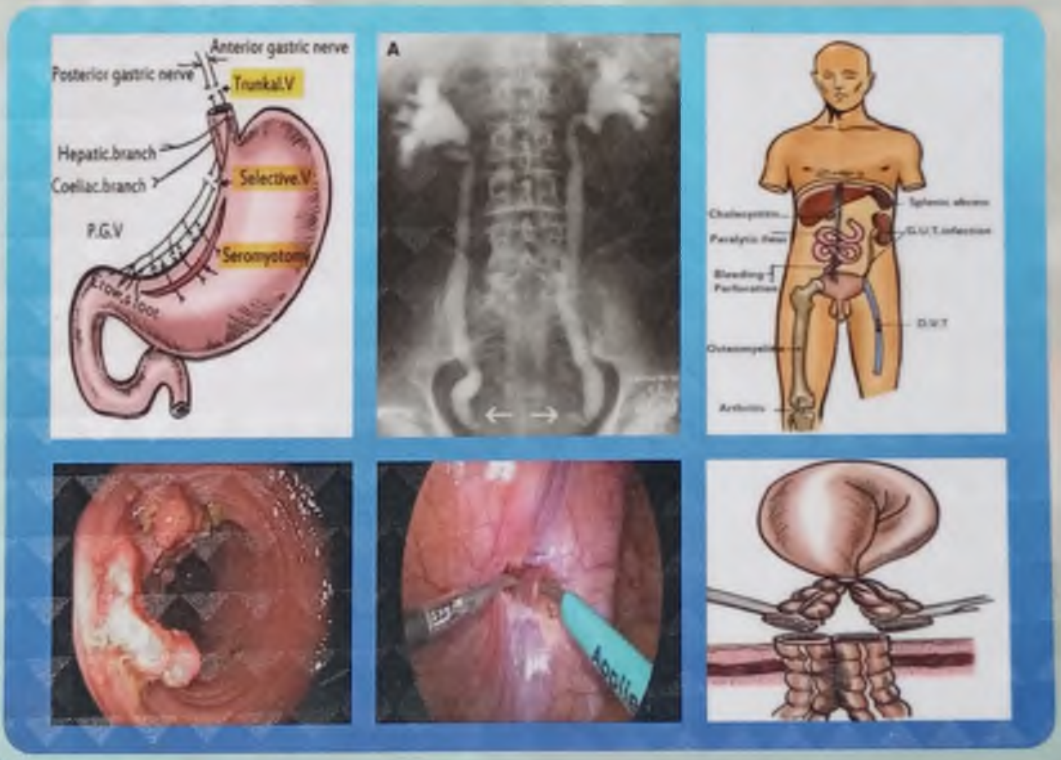
Illustrated Surgery For Undergraduates

By Staff members of General Surgery department
Faculty of Medicine - Zagazig University

Editors

Prof. **Ahmad Kamal** Prof. **Wael Lotfy**



Part 2

First Edition 2018

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Part II

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Chapter IX

Thoracic Surgery

I- CHEST INJURIES

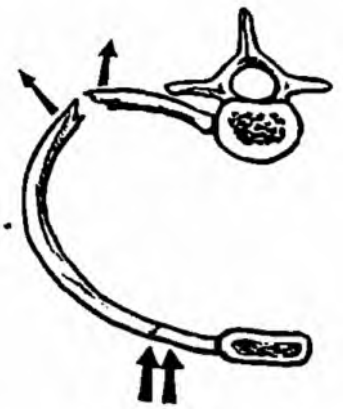
I- Rib fractures

Aetiology: Trauma

1-Direct trauma: fracture at the site of trauma .The fracture ends are driven inward with possible visceral injuries .

2-Indirect trauma: due to anteroposterior compression of the chest (runover accidents). The rib is fractured at the weakest point which is the angle of the rib. Visceral injury is rare as the fractured ends burst outwards.

3-Avulsion trauma: due to forceful muscle contraction during sneezing, coughing may fracture ribs especially in elderly.



Indirect trauma

Pathological Types:

1-Isolated rib fracture

Fracture of the rib at one point.

Aetiology: usually due to direct trauma.

Complications:

1- Limitation of respiratory movement.

2- Pneumothorax, haemothorax, & haemopneumothorax can occur without fracture in children.

3- Lung injury: haemoptysis & surgical emphysema.

Clinical picture

1- History of trauma (may be absent) followed by pain which is increased by coughing or deep breathing.

2- Tenderness at the site of fracture.

3- Crepitus is usually present but it is painful & risky to elicit.

4- Picture of complications.

Investigations:

-Plain X-ray chest: to confirm diagnosis and detect complications.

Treatment:

1- Treatment of the serious complications: e.g. haemothorax has the priority.

II-Treatment of the fracture:

The aim is not to fix the fracture but to relieve pain by:

1-Strapping: Strap the affected side by adhesive plaster, but this limits the chest movement and predisposes to hypostatic pneumonia due to retained secretions.

2- Intercostal nerve block: Injection of local anaesthetic around the neck of the rib.

III-Rehabilitation:

By respiratory exercise (inflating balloons) to open collapsed alveoli and coughing to clear airway & get rid of secretions.

2- Flail chest (Stove in chest)

Definition: Multiple ribs (at least four) fractured at two or more points.

Aetiology: Usually indirect trauma.

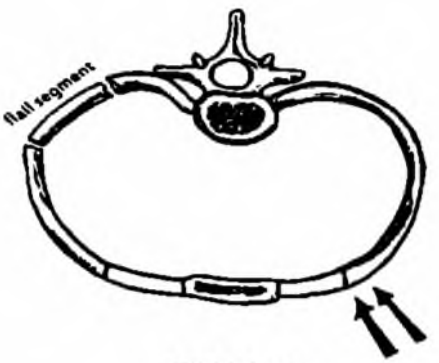
Pathology:

The flail segment is free at its both ends, so it moves independent from the chest wall leading to

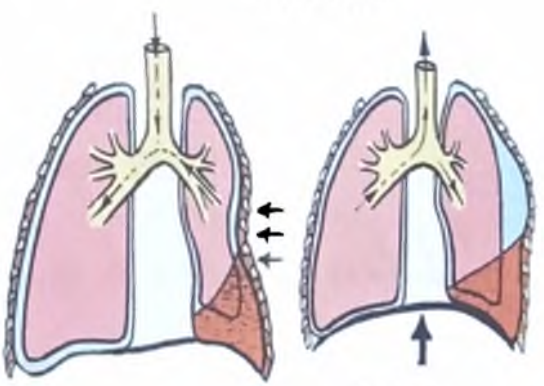
1-Paradoxical movement: The affected lung collapses during inspiration and expands slightly during expiration due to the paradoxical movement of the flail segment.

2-Cross ventilation: the air is re-breathed between the two lungs with hypoxia and retention of CO_2 in the blood.

3-Mediastinal flutter: the paradoxical movement of the affected lung leads to oscillation (pendular movement) of the mediastinum from side to side. This movement interferes with venous return and cardiac action.



Flail chest



Cross ventilation in flail chest

Complications:

Like simple isolated fractured rib +

1- Hypoxia & hypercapnea (Respiratory failure).

2- Shock due to mediastinal flutter (low venous return).

Clinical picture:

Like isolated rib fracture with marked dyspnea, tachycardia possibly with shock and cyanosis

Treatment:

I- Management of multiple injured patient according to ATLS (see trauma)

II- Immobilization of the flail segment (urgent) by:

1- Strapping over a pad of cotton.

2- Musculo-fascial traction using towel clips attatched to weights (skeletal traction).

3-Application of **I**ntermittent **p**ositive **p**ressure **B**reathing **(IPPB)** to serve as internal stabilization of the flail segment. It is applied through endotracheal intubation, but if the period required for artificial ventilation is more than 10 days, tracheostomy is done to avoid laryngeal stenosis secondary to prolonged intubation also tracheostomy decreases the dead space by ¼ and helps easy suction of secretions.

III- Rehabilitation: as before.

II- Pneumothorax

Definition:

Accumulation of air in the pleural cavity.

Aetiology:

I- Spontaneous: due to rupture of emphysematous bullae, cyst or cavity.

II- Traumatic: due to trauma to the chest wall or lung.

III-Artificial: Pneumothorax may be induced in treatment of pulmonary TB. (obsolete)

Pathology:

Types:

1-Closed (simple) pneumothorax

Aetiology:-

Entry of limited amount in the pleural cavity usually through lung tear.

Pathology

The lung collapses with variable degrees, but air is gradually absorbed with spontaneous cure.

Treatment

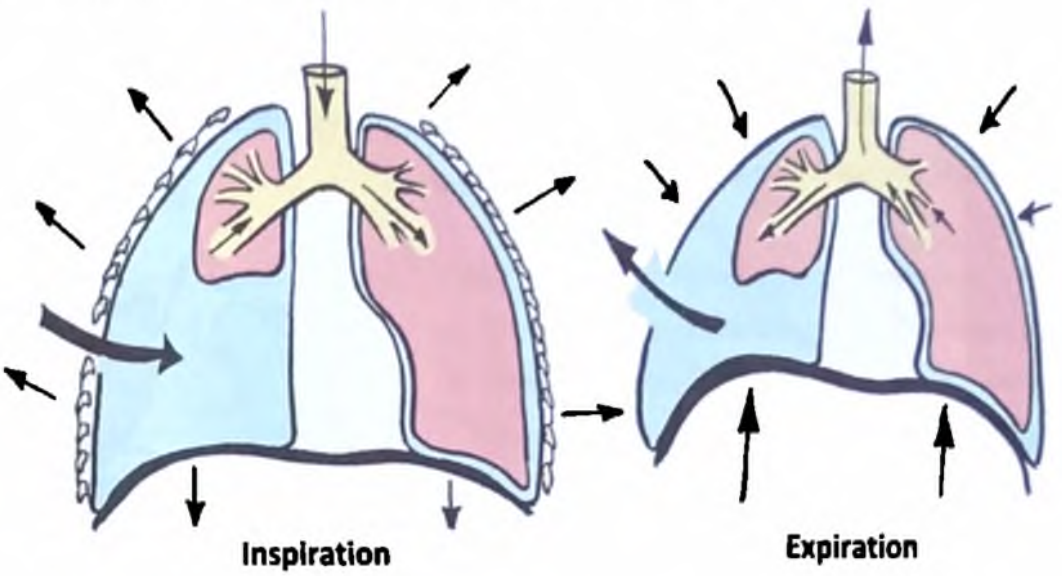
I-Mild cases: asymptomatic with no treatment.

II-Severe cases: with severe dyspnea are treated with application of intercostal tube in the 5th intercostal space above upper rib border and connected to underwater seal for 24 h.

2-Open pneumothorax

Aetiology:

Penetrating trauma makes the pleura communicates with the exterior.



Pathology:

The wound allows free entry & exit of atmospheric air into & out the pleural cavity during inspiration and expiration respectively .This results in paradoxical respiratory movement of the affected lung with cross ventilation between the two lungs & mediastinal flutter (see before).

Complications:

-Respiratory failure (hypoxia & hypercapnea).

-Shock due to mediastinal flutter.

Clinical picture:

-History of trauma (penetrating).

-Sucking wound in the chest may be evident

-The patient is markedly dyspnic, tachypnic with shock, cyanosis & tachycardia.

Treatment

I- Management of multiple injured patient according to ATLS (see trauma)

II-Urgent closure of the sucking wound with pad of vaseline gauze which is strapped firmly to the chest along three borders leaving the fourth border free to act as a valve that gets rid of air inside the pleura during expiration and doesn't allow it to get in again during inspiration.

-After correction of shock and other serious complications the wound is trimmed and closed perfectly with application of intercostal tube as before.

3-Tension pneumothorax

Definition:

Accumulation of air under pressure in the pleura.

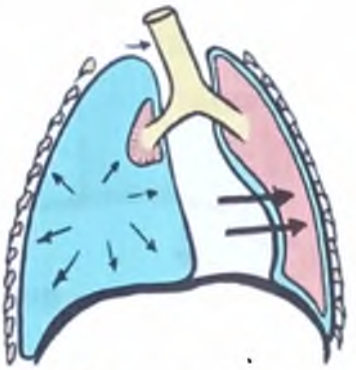
Aetiology:

-Valvular wound allows entry of air into the pleura during inspiration but prevents its escape during expiration, with progressive trapping of increased amount of air in the pleura.

-The valvular wound is either in the lung or chest wall.

Pathology:

There is progressive accumulation of air in the pleural cavity that collapses the ipsilateral lung and displaces the heart and mediastinum to the other side compressing the contralateral lung and interfering with venous return.



Tension pneumothorax

Complications

-Serious respiratory failure.

-Shock due to impeded venous return and heart work.

Clinical picture:

-History of chest trauma.

-Generally: the patient is severely dyspneic with cyanosis, shock, & tachycardia.

-Locally: *Inspection: bulging of the affected side with diminished or absent movement.

*Palpation: tracheal shift to the the other side.

*Percussion: tympanitic hyper-resonance on the affected side.

*Auscultation: diminished air entry on the affected side.

Treatment:

I- Management of multiple injured patient according to ATLS (see trauma).

2-**Urgent** insertion of short wide bore needle in the 2nd intercostal space. It is then replaced by intercostal tube in 5th space connected to underwater seal till full expansion of the lung.

3-Failure of expansion indicates bronchopleural fistula that needs thoracotomy & bronchial repair.

III-Haemothorax

Definition

Accumulation of blood in the pleural cavity.

Aetiology :

1-Traumatic: due to blunt or penetrating trauma causing injury to:

a) Intercostal or internal mammary vessels (commonest source).

b) Lung: bleeding is usually minimal due to low-pressure pulmonary circulation and due to compression of the vessels by the collapsed lung.

c) Major blood vessels (fatal).

2-Iatrogenic: haemothorax may complicate:

a) Thoracic surgery. b) Thoracoscopy. c) Artificial pneumothorax.

3-Spontaneous: haemothorax may complicate:

a) Pulmonary T.B. b) Lung tumours. c) Leaking aneurysm.

Pathology:

Blood is never absorbed spontaneously. If it is not drained, it passes into the following sequel:

1-*Defibrination*: The respiratory & cardiac movements defibrinate the blood so, it remains fluidy for a long time.

2-*Clotting*: the blood stimulates exudation of excess amount of protein–rich effusion with deposition of fibrin on the pleural surfaces & clotting of blood.

3-*Organification*: the deposited fibrin is transformed into a fibrous layer that interferes with expansion of the lung and movement of the chest wall ending in deformity of the chest with crowding of ribs.

4-*Infection*: blood is an excellent culture for bacteria that may cause empyema thoracis.

Complications:

1-Shock: hypovolaemic if severe.

2-Infection: empyema thoracis.

3-Frozen chest: due to organification or empyema.

Clinical picture:-

-History of trauma (absent in spontaneous type).

-Generally: - Manifestations of shock (hypovolaemic).

-The patient is usually dyspneic with chest pain.

-Locally: Inspection: Diminished movement on the affected side.

Percussion: Stony dullness on the affected side with its upper border rising to axilla.

Auscultation: Diminished air entry on the affected side.

Investigations:

1-Plain X ray chest:

-Only obliteration of the costophrenic angle: if the amount is < 500 ml.

-Opacity with its upper border rising to axilla if the amount is > 500 cc.

-Opacity with transverse air fluid level in haemopneumothorax.

2-Intercostal aspiration:

It reveals blood.

Treatment:

I- Management of multiple injured patients according to ATLS (see trauma).

-IV fluids. - Blood transfusion. - O_2 inhalation. -Coagulants (vit k).

II-Definitive treatment:

-*Mild cases:* repeated daily aspiration till clearance and full lung expansion.

-Moderate cases (> 1.5 liter) insertion of intercostal tube usually in the 5th space connected to underwater seal.

-*Severe cases:* → thoracotomy.

Under water seal

Indications of thoracotomy:

1-Severe bleeding > 200ml /hour.

2- Drainage of 1.5 liter or more at insertion of the tube.

3- Persistent bleeding.

4- Associated intrathoracic injuries needing surgery.

Thoracotomy is done usually in the 5th or 6th intercostal space and the bleeding vessels or lung tears are dealt with.

Clotted haemothorax

1-Fibrinolytic therapy: injection of streptokinase 200.000 units in the pleural cavity may liquify the clot that is aspirated after 24 hours. Injection may be repeated if needed.

2-Surgery (thoracotomy): evacuation of clot and decortication of the lung and chest wall

IV- Lung injury

-Usually with minimal bleeding due to low pressure inside the pulmonary circulation.

-Treatment is usually conservative.

-If thoracotomy is done and revealed small lung tear it is sutured or stappled.

-Large lung lacerations may need segmentectomy, lobectomy or even pneumonectomy.

V- Mediastinal injuries

(Cardiac, esophageal, tracheal & big vessels injury)

They are usually serious and need immediate exploration & repair. Cardiac injuries (stab, contusion and cardiac tamponade) are the most serious and associated with profound shock

Treatment:

I- Management of multiple injured patients according to ATLS (see trauma)

-Pericardial tamponad needs: urgent aspiration (see before)

II-Definitive treatment:

-Immediate exploration & repair of myocardial, esophageal, tracheal, & big vessels tears.

VI-Thoraco-abdominal injuries

-*Diaphragmatic injures*: may lead to diaphragmatic hernia or diaphragmatic paralysis with subsequent paradoxical movement of the diaphragm.

-Injuries of the liver on the right side.

-Injury of the spleen and /or stomach on the Lt. side.

Treatment

I- Management of multiple injured patient according to ATLS (see trauma)

II- Definitive treatment:

They need exploration through thoraco-abdominal incision and repair of thoracic, abdominal & diaphragmatic injuries.

N.B. Tear in the dome of the liver may be repaired through the right thoracotomy incision.

VII- Foreign bodies

-Foreign bodies may be inhaled or introduced through penetrating trauma.

-Inhaled foreign bodies should be removed by bronchoscope.

-Penetrating foreign bodies > 1.5 inches should be removed through thoracotomy to avoid infection & abscess formation.

Indications for thoracotomy in chest injury

1- Uncontrollable pneumothorax.

2- Haemothorax, if

-bleeding > 200 ml/ hour, or more than 1500cc.

-persistent bleeding.

-late clotted haemothorax with organization.

3- Mediastinal injuries.

4- Thoraco-abdominal injuries.

5- Foreign bodies > 1.5 inches.

II- Empyema Thoracis

I- Acute empyema

Definition

Pus in the pleural cavity.

Etiology:

I- Causative organisms

1-Gm + Ve (the commonest): Pneumococci, Streptococci & Staphylococci.

2-Gm -Ve: E. coli, Proteus, Pseudomonas.

II- Route of spread:

A- Direct spread:

-From outside through open wounds of the chest.

-From nearby structures:

a- Lung: pneumonia, lung abscess, or carcinoma.

b- Rib: osteomyelitis.

c- Esophagus: perforation or carcinoma.

d- Infra diaphragmatic : subphrenic abscess & liver abscess.

B- Blood born: rare

Pathology:

Types:

I- According to the causative organism:

1-Pneumococcal empyema

-It is a common variety and occurs **after** lobar pneumonia (metapneumonic).

-Pus is thick, & greenish.

-Localization & adhesions are common & early.

-The patient is not so ill.

2-Sterptococcal empyema

-It is less common type that coincides **with** streptococcal bronchopneumonia (synpneumonic empyema).

-The pus is thin & yellowish.

-Localization & adhesion don't occur.

-The patient is seriously ill.

3-Staphylococcal empyema

-It is usually due to direct spread from staphylococcal pneumonia or follows septicemia

4-Putrid empyema

-It is due to rupture of lung or subphrenic abscess into the pleura cavity.

-The infection is mixed one.

II- According to the site:

1-Total empyema: involving the whole pleural cavity.

2-Encysted empyema: localized into pleural recesses (basal, interlobar, apical & mediastinal).

Complications

I- General: Toxemia, septicemia, & pyaemia.

II- Local

1- Chronicity: due to inadequate drainage or presence of broncho-pleural fistula.

2-Empyema necessitatis: A pointing empyema through an intercostal space that may rupture with spontaneous cure without necessity for operation.

3-Extension to the surrounding structures, mediastinum, spine etc....

Clinical Picture

-General:

Constitutional manifestations: Fever (may be rigor), headache, anorexia, malaise & toxaemia.

-Local:

-Inspection: limited movement on the affected side.

-Palpation: may be shift of trachea to the other side.

-Percussion: stony dullness with upper border rising to axilla.

-Auscultation: diminished air entry on the affected side.

-Manifestations of complication.

Investigations

1-Plain X-ray: It shows

-Homogenous opacity obliterating the costophrenic angle with rising border to the axilla.

-Picture of the cause e.g. pneumonia.

2-Aspiration:

To confirm the diagnosis and specimen for culture & sensitivity.

Differential Diagnosis:

From pneumonia, lung abscess, and pneumothorax.

Treatment:

I-General measures

Antibiotics, vitamins, tonics, antipyretics & nutrition.

II Local measures (Pus has to pass)

A- Aspiration

-Repeated aspiration through a two-way tap connected to a needle inserted in the 7th space in the midaxillary or the 9th space in the scapular line.

-Drainage should be:

1- Under complete aseptic conditions.

2- Air tight (two-way tap) to avoid pyopneumothorax which is difficult to manage.

3- As complete as possible.

4- Repeated when needed, i.e. pus re-accumulates.

5- Ended by antibiotic injection.

B- Closed drainage:

-Indicated in:

1- The pus is thick to be aspirated by a needle.

2- Bilateral cases.

-It is equal to repeated aspiration.

-It is done by insertion of an intercostal tube in the 6th space connected to under water seal.

C- Open drainage:

-It is indicated only when full localization has occurred.

-Drainage is done by resection of 2 inches of the overlying rib under local anesthesia then the cavity is opened through the rib bed. The pus & debris are evacuated and a wide bore tube is inserted in the cavity for irrigation.

***NB** No fear of entrance of air as the cavity now is shut off the pleural cavity.

II- Chronic empyema

Definition

It is a chronic abscess in the pleural cavity that prevents the lung from expansion even after drainage of pus.

Actiology:

I- Improper treatment

A- Improper drainage

1-Time: Too early drainage may intodce air or spread infection

Too late drainage → rigid pleura prevents expansion of the lung.

2-Site: -Too high drainage → independent.

-Too low drainage → blocked by diaphragm.

3-Duration: Too short duration→ insufficient

Too prolonged undue drainage may help sinus formation

B- Lack of respiratory exercise:

Failure of lung expansion.

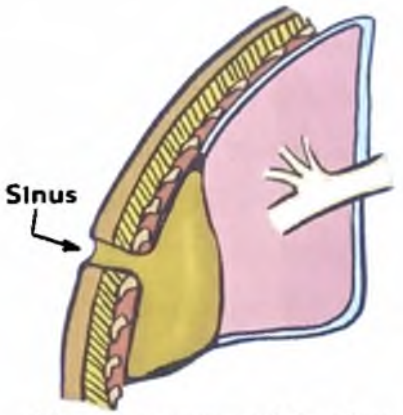
II- Persistent cause:

1-Specific pathology:

Lung T.B, actinomycosis, bronchiectasis, & CA.

2-Persistent source:

Osteomyelitis of a rib & broncho-pleural fistula.



Chronic empyemawith a sinus

Pathology:

Types:

1-Closed empyema: (early chronic empyema):

-It is due to inadequate treatment of acute empyema i.e. with antibiotic only without drainage.

-The organisms may die with their enzymes and the pus is encysted in the pleura.

-Pus may point into a bronchus leading to development of broncho-pleural fistula.

2-Open empyema (late empyema with a sinus):

-It is due to inadequate drainage of acute empyema.

-There is a sinus in the chest wall discharging pus intermittently.

Complications:

I- General:

1-Toxaemia: loss of weight. 2- Secondary amyloidosis.

II- Local:

-Broncho-pleural fistula.

-Deformity of the chest (crowding of the ribs).

-Scoliosis of the spine.

-Respiratory dysfunction especially if bilateral.

Clinical Picture:

I- History of acute empyema which is badly managed.

II- General:

-Toxemia & bad general conditions.

-Clubbing of fingers & may be organomegaly (amyloidosis).

III – Local:

-In most cases, there is a sinus in the chest wall discharging pus intermittently.

-*Inspection:* retraction & limitation of movement on the affected side.

-*Palpation:* shift of the trachea to the affected side.

-*Percussion:* localizes the site of pus.

-*Auscultation:* diminished air entry on the affected side.

Investigations:

1-Chest X ray:

1) Localized collection
2) Crowding of ribs.
3) Elevation of the diaphragm
4) Shift of mediastinum to the affected side
5) Scoliosis of the spine towards the affected side

2- Lipidol pleurogram:

To detect broncho-pleural fistula.

3-Methylene blue or Sudan III test:

Injection of small amount of one of these substances in the pleura may be followed by their expectoration with sputum in case of broncho-pleural fistula.

4- Investigation for the cause:

Sputum analysis, bronchoscopy, CT, & pleural biopsy.

Treatment:

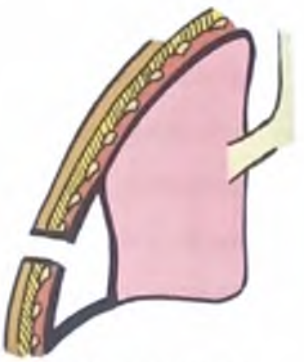
I- General measures

As before + treatment of the cause.

II- Local measures

A-Again drainage:

But in the proper site and for the proper time together with proper antibiotic & physiotherapy may help re-expansion of the lung and resolution.



Open drainage

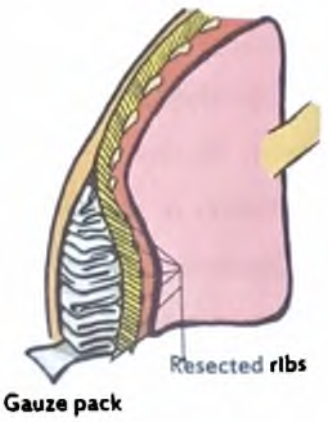
B- Decortication:

It means removal of thickened visceral pleura over the lung to help its expansion by positive pressure ventilation.

C- Thoracoplasty (not done nowadays):

-Obliteration of empyema cavity by collapsing the thoracic wall or by use of muscle flap.

-It gives marked deformity of the chest wall & doesn't improve the lung function.



Thoracoplasty

Surgical treatment of pulmonary TB.

Indications

Surgical treatment must be considered only when medical treatment fails to control the disease.

Procedures:

Surgical treatment includes two groups of therapy

I- Collapse therapy

The aim is to help closure of T.B cavities by relaxing the lung tissue around. It is either

A- Temporary collapse: by

1- Artificial pneumothorax.

2- Artificial pneumoperitoneum (obsolete)...

3- Extrapleural pneumothorax: introduction of air outside the parietal pleura to cause selective collapse of the diseased segment.

4- Phrenic nerve crushing: The nerve recovers in 4-6 months.

B- Permanent collapse, by

1-Phrenic never avulsion: The nerve never recovers.

2-Exrapleural Plombage: Extrapleural introduction of synthetic material.

3-Thoracoplasy: The idea is to remove the ribs overlying the diseased segment to induce localized collapse. It was done in two or more stages. In the first stage, the 3rd, 2nd & 1st ribs are removed subperiosteally, usually accompanied with separation of the lung from the parietal pleura (apicolysis) to help collapse. In the 2nd stage, more ribs are removed.

II- Resection

Indicated in:

1- Solid TB. lesion (tuberculoma).

2- Large thick cavities resistant to collapse therapy.

3- TB. bronchiectasis.

4- TB. empyema with permanent collapse.

5- Completely destroyed lung with multiple cavities.

Resection includes segmentectomy, lobectomy, pneumonectomy, and pleuro-pneumonectomy.

Complication of surgical treatment:

1- Spread of the disease.

2- Atelectasis.

3- Bronchopleural fistula.

4- Empyema thoracis.

III-Tumours

I-Benign tumours

Benign tumours represent only 1-2 % of all tumours of the lung

Pathology:

Types:

1- Bronchial adenoma

-It arises from the mucosa of one of the main bronchi.

-It grows both in the lumen and into the adjacent lung tissue assuming an *hour-glass shape* and hence it called *dumbbell tumour*. So it is considered local malignant tumour or low grade malignancy (metastasis are recorded in 15 % of cases)

-Histologically, it resembles mixed salivary tumours .

2-Hamartoma:

It is a tumour –like condition composed of collection of tissue normally found around the tumour but haphazardly arranged. So it normally contains respiratory epithelium, cartilage ,fat, blood vessels and muscles

3-lipoma,fibroma,angioma and chondrromas are rare and now are considered hamartoma.

Complications:

1-haemopytsis

2-collapse (atalectasis)

3-infection,bronchiactasis and lung abscess.

Incidence:

They are common in young adult individuals with no sex prediction

Clinical picture :

- They may be a symptomatic for a long time
- Longstanding cough not responding to treatment.
- Symptoms of complications

Investigations:

1-Plain X ray : it may show the tumour calcification or complications such as atelectasis.

2- Bronchoscopy: it shows the tumour bronchial adenoma appears as rounded smooth like tumour while hamartoma appears as pale firm swelling.

3-CT scan of the chest

Differential diagnosis:

From other solitary pulmonary shadow

Treatment:

Local excision if small or lobectomy if associated with permanent lung damage

Malignant tumours

Bronchogenic carcinoma

Aetiology:

I-Predisposing factors:

1-Cigarette smoking is undoubted risk factor

2-Inhalation of certain irritant such as arsenic, silica, cobalt and fumes of petrol in industrial workers and in crowded cities

Pathology:

Site: bronchogenic carcinoma has two main types according to its site

1- Central type (75%)

-It arises from main bronchus or one of its main divisions. It leads to bronchial obstruction, atelectasis, bronchiactesis or lung abscess.

2-Peripheral type 25%

-it arises from smaller bronchi

-it may remain symptomless for along time

-Pancoast tumour belongs to this type

MAC

It usually gives an irregular hard mass that grows both into the lumen obstructing bronchi and to the surrounding tissue. Cut section shows areas of hage., necrosis and occasionally cavitations.

MIC :

there are the following types

1-Squamous cell (epidermiod) carcinoma (60%)

-It trends to occur centrally.

-It is composed of undifferentiated arranged squamous cells.

2- Adenocarcinoma :

-It has the following subtypes

a-Acinar :Acini lined with columnar cells.

b-Papillary: Cystic areas with papillary projections

c-Alveolar :from terminal bronchioles.

3- *Small cell (oat-cell) carcinoma* (15%)

-It is composed of undifferentiated small cell with very scanty fibrous stroma

-It usually occurs near the hilum .

-It is the most malignant variety.

4- *Large cell carcinoma*:

- It is composed of large cells with cytoplasmic deposits and occasional giant cells

- It is commonly peripheral.

5- *Other types*

❶ Adenoid cystic carcinoma

❷ Mucoepidermiod (adenosquamous)carcinoma

❸ Carcinoid tumour :central, slow growth ,late metastasis and may cause carcinoid syndrome.

Spread:

1- Local spread:

a- Intrinsic spread to the lumen of the bronchus with obstruction & to the nearby lung tissue.

b- Extrinsic spread to the mediastinum, pleura, pericardium and the cheat wall.

2-Lymphatic spread: to hilar, tracheo-bronchial and lower deep cervical LNs.

3- Blood spread: mainly to the liver, adrenal, brain and bones.

Complications:

1- Bronchial obstruction that leads to atelectasis, bronchiactasis, lung abscess.

2- Haemoptysis.

3- malignant pleural effusion, haemothorax and empyema.

4- Mediastinal syndrome due to enlarged LN(s)

5- Thoracic outlet syndrome : pancost tumour arising from the apex of the lung invading the lower trunk of bracial plexus leading to pain in the upper limb & Horner's syndrome with erosion of the upper two or three ribs .

6- Paramalignant syndrome :

It means systemic manifestations due to malignancy not caused by metastasis (systemic non metastatic manifestations). it is attributed to hormones or other substances elaborated from the primary tumour and disappear after successful excision of it. It includes

❶ Cushing syndrome (due to ectopic ACTH

❷ hypercalcemia (parathormone like polypeptide)

❸ water retention (ADH)

❹ carcinoid syndrome (serotonin 5HT)

❺ tender gynaecomastia (ectopic gonadotropin)

- ❻ malignant neuropathy :myasthenia, dermatomyositis, peripheral neuritis
- ❼ pulmonary osteoarthropathy is characterized by clubbing of fingers ,joint pain & oedema.
- ❽ migration thrombophlebites (Trausseau sign)
- ❾ arterial thrombosis.
- ❿ endocarditis

7-Infilteration of intercostal nerves leads to severe chest pain and insomnia

8- Infilteration of phrenic nerve leads to diaphragmatic paralysis

9- Infiltration of the recurrent laryngeal nerve leads to hoarseness of voice

10- Distant metastasis

Incidence:

-Bronchogenic carcinoma is the commonest carcinoma in the world

-It is the commonest malignancy in male and the second commonest in female

- It is much commoner in smokers than non smokers

-It is common in the middle age

Clinical picture:

The presentation of bronchogenic carcinoma is quiet variable from patient to another. There are the following types

1- Insidious type (the usual type)

The patient complains of dry cough ,usually attributed to smoking, that later becomes asthmatic then associated with dyspnea ,chest pain & haemoptysis.

2- Acute type sometimes the onset is acute with fever ,pneumonia that is resistant or relapse within few days or weeks.

3-Latent type:

The condition remains silent for a long time till it is discovered accidentally on routine X ray examination or the patient complains of vague general symptoms like Anaemia, asthenia and anorexia. The diagnosis may be delayed till distant metastasis present.

4- Pancoast type :presents with pancoast syndrome (see before)

5- Extra-pulmonary type: patient presents with paramalignant syndrome. (see before)

Investigations:

1-Plain chest X ray :it may show area of consolidation atelectasis or hilar shadows

2-Sputum cytology for exfoliated malignant cells

3-Bronchoscopy it reveals the lesion and allows punch biopsy

4-Needle biopsy under fluoroscopy or CT guidance can establish the diagnosis in 60 %of cases especially in peripheral lesion

5-Mediastinoscopy to allow for mediastinal LN biopsy

6- CT chest :of extreme importance

7- scalene LN biopsy.

8- Other investigation to assess for spread of all the tumour e.g. bone scan

Differential diagnosis:

1- From other causes of haemoptysis

2- From other causes of unresolving or recurrent pneumonia

3- From other causes of solitary pulmonary nodules (coin shadow in plain chest x ray)

a) 35% primary carcinoma b) 35% non specific grauloma c) 20 % tuberculoma

d) benign tumours 5% e) 5% secondary carcinoma.

Treatment:

A- inoperable case

*One third of cases are considered inoperable on the first diagnosis

*Another third is founded to be inoperable after exploration

Criteria of inoperability

1- surgically unfit patient

2- signs of distant metastasis (not paramlignant syndrome)

3-involvement of LN(s) not included in radical resection e.g. cervical (scalene LN)

4- incompletely resectable tumour

a-tumour infiltrating chest wall or diaphragm.

b-tumour invading the trachea or oesophagus or pericardium

c-tumour infiltrating the recurrent laryngeal nerve

-Inoperable cases are treated with

1-Palliative resection: in resectable cases to relieve or avoid local complication such as haemoptysis or infection and relieve paramlignant syndrome

2-Radiotherapy to relieve SVC obstruction ,bronchial compression and pain of bone metastasis

3-Chemotherapy : cytotoxic drugs and antibiotics for infection

B- Operable

* Only one third of cases is operable

Radical pneumonectomy removal of the lung with hilar and mediastinal LNs. Lobectomy may be advised in peripheral carcinoma in old patients

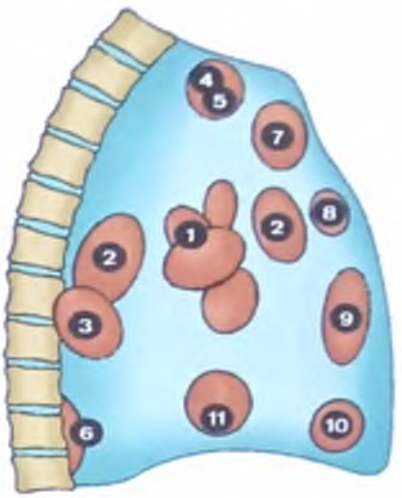
Prognosis

-The 1- year and 5-year survival rates are 20 % and 8% respectively

-The overall mean survival is about 9 months

Mediastinal masses

1. Hilar lymph nodes: sarcoidosis, TB, lymphoma & metastases.
2. Aortic aneurysm.
3. neurogenic tumors: Neuroblastoma, neurofibroma
4. Bronchogenic cysts.
5. Enterogenous cysts.
6. Paravertebral abscess.
7. Retrosternal goiter.
8. Thymic masses: thymoma.
9. Teratoma and dermoid cysts.
10. Pericardial cysts.
11. Hiatus hernia



Clinical picture:

I-Asymptomatic in 30 to 50 % of cases and would be discovered accidentally on chest X ray

II- Symptomatic cases (mediastinal syndrome): due to compression on mediastinal structures

- Tracheal compression: wheeze, and breathlessness.
- Esophageal compression: dysphagia.
- Superior vena cava compression: congested neck veins.
- Cardiac compression: tamponade.
- Nerve compression: hoarseness, and Horner's syndrome.

Investigations:

1. Postero-anterior chest radiograph will diagnose the problem.
2. Lateral chest radiograph will locate the compartment affected.
3. CT and MRI.
4. Mediastinoscopy, bronchoscopy, and eosophagoscopy.
5. Biopsy.

Treatment:

According to the cause.

N.B. 95% of asymptomatic masses are benign, while 50% of symptomatic cases are malignant.

Chapter X

Abdominal Surgery

Anterior abdominal wall

The anterior abdominal wall is formed of the following layers:

1-Skin.

2-Subcutaneous tissue
- Fatty layer = (*Camper's fascia*)
- Membranous layer = (*Scarpa's fascia*)

3-Muscle layers:

(External, internal oblique & transversus abd. muscles).

4-Fascia transversalis.

5-Parietal peritoneum.

Muscles of the anterior abdominal wall

1-External abdominal oblique muscle:

Origin: From the lower eight ribs interdigitating with serratus anterior & latissmus dorsi muscle.

Insertion: Its fibres are directed downward, forward & medially to be inserted in:

1-linea alba (through its aponeurosis).

2-Pubic crest.

3-Pubic tubercle.

4-Anterior superior iliac spine (ASIS).

5-Iliac crest (outer lip).

-From the ASIS to the pubic tubercle, the ext. oblique aponeurosis is stretched, free and folded backward to from *the inguinal ligament*

External oblique muscle

2-Internal abdominal oblique muscle:

Origin: from

1- Lumber fascia.

2- Iliac crest (intermediate area).

3- Lateral 2/3 of the inguinal ligament.

Insertion: its fibres are directed upward forward & medially to insert into:

1-The lower 6 ribs or their costal cartilages.

2-linea alba

2-With the transversus abdominis into the pubic crest & pectineal line (conjoint tendon)

Internal oblique muscle

3- Transversus abdominis muscle:

Origin: from

1-lower 6 ribs or their costal cartilages;

2-lumber fascia.

3-Iliac crest (inner lip).

4-lateral 1/3 of the inguinal ligament.

Insertion: Its fibres pass horizontally to:

1- Insert into linea alba.

2-fuse with internal oblique in their lowest fibres to form the conjoint tendon that inserts into the pubic crest & pectineal line.

Transversus abdominis muscle

4-Rectus abdominis muscle:

Origin: from the pubic crest.

Insertion:

-Its fibers are directed upward to insert into the xiphoid cartilage and into 5th, 6th, 7th costal cartilages.

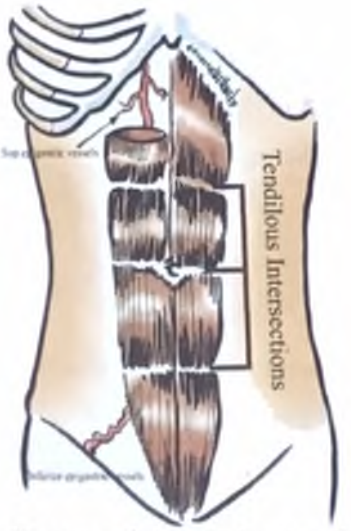
-The rectus muscle is interrupted with 3 or 4 tendinous intersections, which are placed as follow:

1-one opposite the xiphoid cartilage.

2-one midway between the xiphoid cartilage and umbilicus.

3-one opposite the umbilicus.

4-one midway between the umbilicus & the symphysis pubis



Rectus abdominis muscle

The rectus Sheath:

It is the sheath formed by fascia & aponeurosis of the abdominal wall enclosing the rectus muscle.

-*Its anterior wall* is formed of:

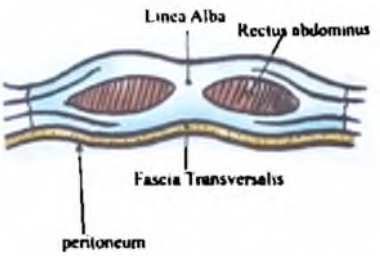
*external oblique aponeurosis.

*anterior lamella of internal oblique aponeurosis.

-*Its posterior wall* is formed of:

*The posterior lamella of the internal oblique aponeurosis.

*The aponeurosis of transversus abdominis muscle.



The rectus sheath

The posterior rectus sheath is missed below the level midway between the umbilicus & symphysis pubis and end sharply with a concave border called (the arcuate line).

Nerve supply of abdominal muscles:

-The lower five inter costal nerves.

-The subcostal, iliohypogastric & ilioinguinal nerves.

-The cremasteric muscle is supplied by the genital branch of the genitofemoral nerve.

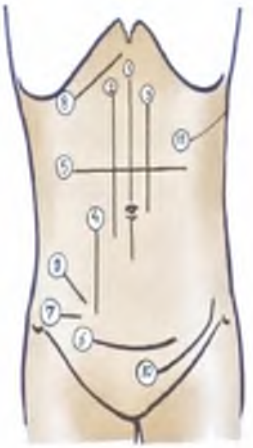
Action:

1-They form the anterior abdominal wall maintaining the abdominal viscera in position.

2- Contraction increases the abdominal pressure and so helpful in coughing, sneezing, micturition, defecation, labour & vomiting.

3-The rectus abdominis muscles flex the trunk forward.

Abdominal incisions
(Laparotomy)



Abdominal incisions

I- Vertical incisions

1-Midline incision ❶

Through linea alba.

**Advantages*

1-Good exploratory to both sides of abdomen.

2-Rapidly made, freely extended & rapidly closed.

**Disadvantage*

Poor healing with high incidence of burst abdomen & incisional hernia.

2- The paramedian incision❷:

1 inch lateral to the midline. The rectus muscle is retraced laterally to preserve its nerve supply then the posterior sheath and peritoneum are opened as one layer.

Advantages

1-No muscle cutting & no nerve injury.

2-The trap door effect of the rectus muscle strengthens the incision and lowers the possibility for burst abdomen & herniation.

3-Can be freely extended.

Disadvantages It takes long time to make with some blood loss.

3- Transrectal incision❷:

Like paramedian incision but the rectus is split longitudinally rather than retracted laterally.

Advantage

It is more rapid than paramedian incision.

Disadvantage

Loss of the trap effect makes the incision weaker

4-Pararectal (Battle's) incision● :

Like paramedian incision but made more laterally and the rectus muscle is retracted medially.

Disadvantages

1-It cannot be extended freely.

2-It denervates part of the rectus.

II- Transverse incisions

1-Transverse epigastric incision●:

It is done midway between umbilicus & xiphoid process by muscle cutting.

Advantages:

1-Good exploratory to all viscera of the upper abdomen.

2-Perfect healing as the abdominal muscles pulls transversely on contraction thus they reinforce the incision rather than disrupt it.

3-The nerve supply to the recti is segmental and not affected.

Disadvantage:

Time-consuming incision with some blood loss.

2- Transverse suprapubic (Pfanstiel) incision●:

It is done over the interspinous crease. Transverse anterior rectus sheath incision, lateral separation of both recti, then transverse incision of peritoneum.

Advantages:

- Good exploratory to pelvic organs (widely used by gynecologist).
- Very good healing power.
- Very cosmetic appearance.

Disadvantage:

- cannot be extended.
-Liable to contamination from perineum.

3- Lanz's incision●:

-It is a modification of Mc Burney's incision for appendicectomy.

-It is a transverse incision over the interspinous line and the deep layers are opened like Mc Burney's incision.

Advantage:

It gives a better cosmetic result.

Disadvantage:

It cannot be enlarged in the vertical direction in case of high-situated appendix.

III Oblique incisions:

1- Kocher's subcostal incision❽:

It is parallel to and one inch below the costal margin extending from the midline to the tip of the 9th costal cartilage.

Advantages:

- It gives good access to gall bladder& biliary system on the Rt. side and spleen. on the Lt. side
- Double Kocher's (Chevron incision) or its modification *bucket handle incision* gives good access to the upper abdomen.

Disadvantages:

-It cannot be enlarged freely.

-It commonly injures the 8th. 9th, 10th intercostal nerves.

1- Mc Burney's incision❾:

-Skin incision (see later).

-The external oblique aponeurosis is cut obliquely with the direction of fibres and the internal oblique & transversus abdominis muscles are split with the direction of their fibres.

-Then the peritoneum is grasped between two mosquito forceps & opened.

Advantages:

1- Gridiron effect supports the incision and prevents herniation.

2- It gives direct access to the appendix.

Disadvantage:

-It cannot be extended freely.

-It may injure the ilioinguinal nerve causing direct inguinal hernia.

3- Abernethy's incision❿:

For stone middle ureter (see later).

The ideal abdominal incision should be:

1-Good exploratory: gives a good access to the proposed organs.

2-Extensible: it could be extended freely in both directions.

3-Easy to be made and easy to be closed with minimal bleeding.

4- Minimally destructive: less injurious to important structures.

5-Strong incision with good power of healing and with good cosmetic results.

The umbilicus

I- Congenital diseases

A- Anomalies of vitello intestinal duct: See later

B-Anomalies of the urachus: See later

C-Congenital umbilical hernia See later

II- Inflammation

Omphalitis

A- Neonatal omphalitis:

-It is inflammation of the umbilical cord stump.

-It is dangerous as it may cause thrombophlebitis of umbilical vein that may end in portal pyaemia, portal hypertension and may be death.

B- Adult omphalitis (umbilical dermatitis)

-It is due to absence of hygiene.

-It may cause umbilical granuloma (granuloma pyogenicum) or umbilical stone (omphalith) due to accumulation of desquamated epithelium with inspissated mass of dirt

-It is treated with repeated cleansing & antibiotic- If granuloma, it should be curetted

C-Pilonidal sinus of the umbilicus

(See later)

III-Umbilical fistulae

A- Faecal fistulae:

1- Congenital (patent vitello-intestinal duct).

2- Tuberculous peritonitis.

3- Postoperative fistula after midline laparotomy.

4- Malignant ulceration of the colon.

B- Urinary fistula:

Congenital due to patent urachus (see urology).

C- Biliary fistula:

Usually due to subacute perforation of acute cholecystitis.

IV – Endometriosis

-It is an ectopic endometrium, common in middle-aged females.

-It causes pain & bleeding at the time of menstruation.

-It is treated with excision of the umbilicus.

V- Hernia (See below)

Abdominal Hernias

Definition:

Hernia is abnormal protrusion of a viscus through a defect in the wall of its containing cavity.

Aetiology:

I-Congenitally preformed sac:-

Only in congenital inguinal hernia, congenital umbilical hernia & cong. diaphragmatic hernia.

II-Acquired: (Defect acted upon by increased intra-abdominal pressure)

A -Defect (weakness of abdominal wall):

1 - *Stretch*: pregnancy, ascites.

2- *Senile muscle atrophy*: due to ↑↑ catabolism.

3- *Obesity*: fat globules separate muscle bundles & aponeurotic fibers thus weaken them.

4- *Nerve injury*: during operations, e.g. illioinginal nerve injury →D.I.H.

B - Increased intrabdominal pressure:

1-Chronic straining: ch. Cough, ch. Constipation, urinary outflow Constriction (BNO. SEP. & stricture urethra).

2-Abdominal masses: Hepato-splenomegaly, pregnancy, ascites....etc.

Pathology:

I- Types: according to the site.

A- External abdominal hernias: (outside the abdominal cavity).

1- Inguinal hernia (direct & indirect) 73%.

2- Femoral hernia (17%).

3- Umbilical hernia (8.5%).

4- Rare types e.g. lumber, epigastric.... etc (1.5%).

These figures do not include incisional hernia, which is the 2nd commonest hernia after inguinal hernia.

B- Internal abdominal hernias (inside the abdominal cavity).

Such as hernia through paraduodenal fossa, mesenteric defect or foramen of Winslow.

II-Structure: each hernia has:

A-Defect: The hole in the wall through which hernia protrudes.

B-Sac: pouch of peritoneum that envelops the protruding viscus.

It has a mouth, neck, body & fundus.

C-Contents: They are the protruding viscus.

Structure

All abdominal organs except the pancreas may contribute to the contents of hernias:

1- Intestines (enterocele). 2- Omentum (omentocele).

3- Part of the circumference of the intestine (Richter's hernia).

4- Meckel's diverticulum (Littre's hernia).

5- Two loops of the bowel (Hernia in W or Maydle's hernia).

6- Ovaries & fallopian tubes. 7- Appendix (amyand's hernia).

Strangulated Richter's hernia

Strangulated Littre's hernia

Maydle's hernia

D- Coverings: The layers that overlay the sac.

E- Descent: It is the direction of protrusion of the contents.

Anatomy of inguinal canal

Inguinal canal is a short tract (4 cm long) running obliquely in the lower part of the abdominal wall from the deep inguinal ring to the superficial inguinal ring.

1-Deep inguinal ring: rounded defect in the fascia transversalis & lies 1/2 inch above the midinguinal point (midway between the anterior superior iliac spine (ASIS) & symphysis pubis (SP).

Superficial (external) inguinal ring: a V shaped slit in the external oblique aponeurosis 1/2 inch above and lateral to public tubercle.

Boundaries of the inguinal canal:

Anterior wall is formed of:

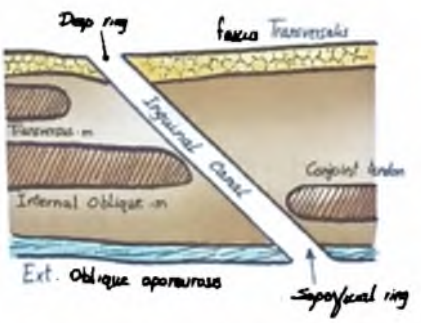
- External oblique aponeurosis along the whole length.
- Internal oblique muscle in the lateral half.

Posterior wall is formed of:

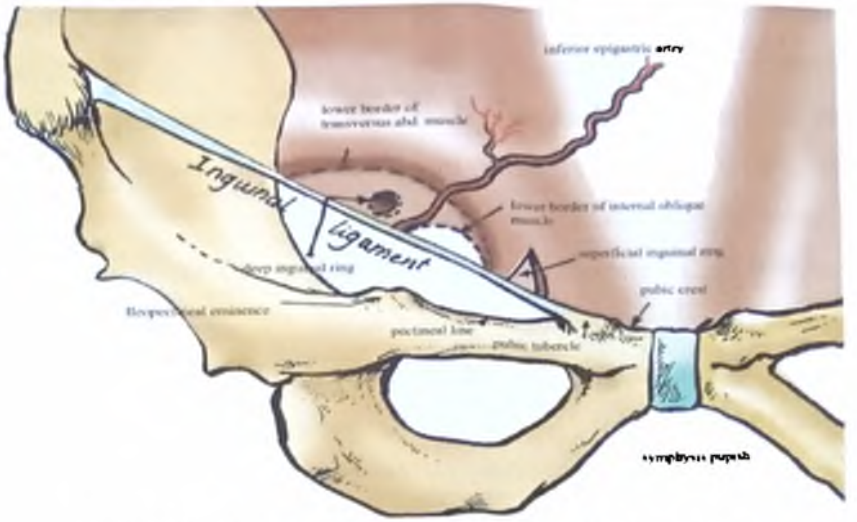
- Fascia transversalis along the whole length.
- Conjoint tendon in the medial half.

Roof: is formed of arching lower fibers of int. oblique.

Floor: is formed of inguinal (Poupart's) ligament.



Boundaries of the inguinal canal



Anatomical landmarks in the inguinal region

Structures that pass through the deep inguinal ring:

1- Spermatic cord in males & round ligament in females.

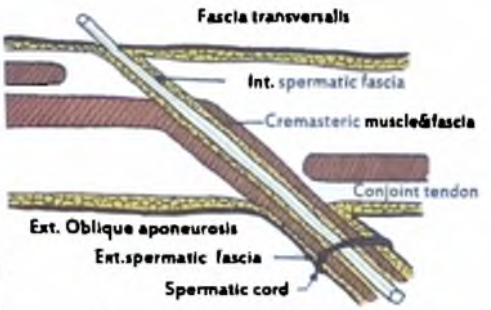
2- Cremastric artery (branch of the inf. epigastric artery).

3- Genital branch of genitofemoral nerve (motor to cremastric muscle).

Structures that pass through the superficial ring.

The previous structures plus:

- Internal spermatic fascia.
- Cremastric muscle and its fascia.
- Ilioinguinal nerve.



Layers of the spermatic cord

**Hasselbach's (Inguinal) triangle:*

-It is bounded by the inferior epigastric artery laterally, the lateral border of the rectus abd. muscle medially and the inguinal ligament inferiorly .

-It represents the weak part of the post wall of inguinal canal.

Hasselbach's triangle

**The internal oblique m. has a triple relation to the cord.*

1- At first it is anterior to it (lat. part of ing. canal).

2- Then it is above it (forming the roof of the canal).

3- Then it descends behind it (medial part of the canal).

**Cooper's lig* = (pectineal ligament)

It is a strong fibrous band stretched over the iliopectineal line on superior border of the pubic ramus.

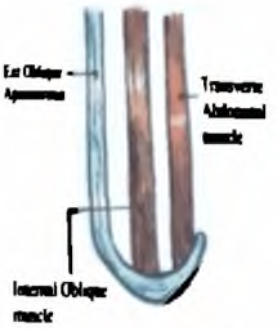
Inferior epigastric artery is a branch of the external iliac artery (The latter gives two branches only, inf. epigastric art & deep circumflex artery).

- The inferior epigastric art. ascends upward medial to the deep ing. ring and deep to fascia transversals to enter the rectus sheath in front of the arcuate line where it anastomoses with the superior epigastric artery .

Ilioinguinal nerve (L1, 2) is motor to the conjoint tendon (at the Mc.Burnny level) and sensory to the scrotum and inner side of the thigh.

- Its injury during appendicectomy leads to paralysis of conjoined T. that causes direct inguinal hernia.
- Its injury during hernia operations. → Only numbness of scrotum.

-Inguinal ligament (Poupart's ligament): It is the lower free border of ext. oblique aponeurosis. It is folded backward upon itself to give origin to internal oblique & transversus abdominis muscle. It is stretched between The ASIS. & public tubercle .



Inguinal ligament

Inguinal hernias

I- Oblique (Indirect) ing. hernia

Definition:

It is an external abdominal hernia that passes through the deep ing. ring ,enters the inguinal canal and may reach the scrotum .

Aetiology:

I- Congenital theory:

Hernial sac is due to persistent processus vaginalis either partially or completely. It opens up when the intra-abdominal pressure increases.

II- Acquired theory:

Hernial sacs are formed due to protrusion of the peritoneum in front of increased intra-abdominal pressure through the deep ing. ring due to deficiency of mechanisms that normally prevent herniation . These mechanisms include:

1- *Valvular mechanism*: Duo to the oblique course of the inguinal canal. Contraction of abdominal muscles will approximate the walls →closure.

2- *Shutter mechanism*: int. oblique muscle forms a semi-sphincter around the cord. When the conjoint tendon contracts, it descends and supports the posterior wall of the canal.

3- *Plugging mechanism*: The cremastric muscle contracts with the abdominal muscles and becomes more bulky plugging the deep ring.

The congenital theory is supported by the following facts:

1-Oblique inguinal hernia especially in first decade is commoner on the right side, as the Lt. testicle descends earlier with more time for the processus vaginalis to close.

2-Herniotomy (removal of the sac) alone is sufficient in children & adults.

3-Cong. patent processus vaginalis was found in autopsy from individuals who had not hernia.

Pathology:

Site: Groin, above the groin crease.

Defect: Stretched deep ring.

Sac: It is anterior to the cord structures. It may be:

1- Congenital: Persistence of the whole processus vaginalis i.e. the tunica vaginalis remains communicating with the peritoneum and the testicle appears as if it is inside the sac.

2- Funicular: Only proximal portion of processus vaginalis persists. The sac ends in the cord.

3- Infantile: Like funicular sac but with redundant tunica that overlaps it.

4- Bubonocele: The sac is limited to the inguinal canal i.e. passes through the deep ring not the superficial one.

5- Complete sac: Longstanding hernia reaches the bottom of the scrotum but the testicle can be felt separate from the contents.

6- Saddle shaped or pantaloon sac: Both direct & indirect hernia coincides & is separated by the inferior epigastric vessels.

7- Sliding hernia (Hernia en glissade): a nearby organ slides with the peritoneum to form a part of the wall of the sac.

Contents: As before, (mention).

Coverings: 1- Skin & superficial fascia.
2-External spermatic fascia (from ext. oblique m.).
3- Cremastric muscle & fascia (from int. oblique m.).
4- Internal spermatic fascia (From fascia transversalis).
5- Extraperitoneal fat.

Descent: Downward, forward and medially (reduction should be in reverse direction).

Complications:

1- Irreducibility 2- Obstruction. 3- Strangulation. 4- Inflammation.

Incidence:

- It is the commonest type of hernia. It represents about 85% of ing. hernia.
- It is more common in males (20: 1).
- It is commoner in the right side (in the first decade of life), bilateral in about 30% of cases.

Clinical picture:

Symptoms:

1-Groin swelling which appears & enlarges on standing, coughing or straining and disappears on lying down.

2- Large hernias may produce dragging pain.

3-In congenital hernia, the swelling appears on crying (history diagnosis from the mother).

Signs:

1- Swelling: has the following characters.

a- In the groin above inguinal lig. (pubic tubercle) .

b- Shows expansile impulse on cough.

c- Reduced in certain direction (upward, backward & laterally) .

2- The contents of the sac can be suspected clinically.

	Enterocele	Omentocele
1- Consistency	Soft (Cystic)	Doughy
2- Gurgling	May be felt during reduction	Absent
3- Reduction	Difficult at the start	Difficult at the end
4- Percussion	Resonant	Dull
5- Auscultation	Intestinal. sound,	Silent

- Ovaries or testis gives visceral pain on pressure.

3-Transillumination: opaque except in infancy due to thin wall of gas containing intestine.

4-Special tests:

a- Internal ring test:

- Let your patient to lie down and reduce the contents.
- Press over the internal ring (where?) with your finger.
- Ask the patient to stand then cough while maintaining pressure on the ring.
- OIH does not descend while D.I.H descends.

b- External ring test: (not preferred --- painful).

-Reduce the hernia.

-While the patient is standing invaginate the neck of the scrotum by the little finger of corresponding hand into the external ring.

-Ask the patient to cough and observe the impulse.

In OIH → Wide ext. ring (normally it admits the finger tip). The impulse of cough strikes the tip of the finger.

In DIH → The ext. ring is normal. The impulse strikes the medial side of the finger.

External ring test

C-The Three fingers test (Zieman's technique) :

-While the patient is standing, stand behind the patient & Place:

1- The index finger over the internal ring.

2- The middle finger over the external ring (Hasselbach'sΔ)

3- The ring finger over the femoral canal.

-Ask the patient to cough and note which finger feels the impulse.

Three fingers test

Investigations:

Mainly to detect possible predisposing factors. Chest X ray, PR and IVU, abd. U/S ...etc.

Differential diagnosis:

A- Bubonocele should be differentiated from other inguinal swelling.

I- *Hernias*: 1-DIH.: Int. ring test & Zieman's test.

2- Femoral hernia, below & lat. to the pubic tubercle.

II- *Lymph node*: Inguinal lymphadenitis → tender multiple swellings.

III- *Vein*: Saphena varix: → compressible swelling at the saphenous opening show thrill on cough & evidence of V.V. of the lower limb.

IV- *Artery*: Iliofemoral aneurysm → pulsating swelling along the line of the artery.

V - *Muscle*: Psoas abscess→ cross fluctuant swelling across the inguinal ligament.

Psoas bursitis → disappear on flexion of the hip & evidence of hip osteoarthritis

VI- *Testicle*: Undescended or ectopic testicle (empty scrotum).

VII- *Skin & SC tissues*: sebaceous cyst, lipoma, fibroma, neurofibroma ...etc.

B- Funicular and complete types from other inguinoscrotal swellings:

I- *Hydrocele*: Congenital, infantile and hydrocele of hernial sac.

II - *Cord*: 1-Diffuse lipoma of the cord.

2- Endemic funculitis.

III-*Veins*: Varicocele,worms like mass on palpation, redundant scrotum, and thrill on cough.

Treatment:

1- Truss: a special appliance used by the patient to prevent descent of the contents into the sac.

Indications:

Only if the patient is unfit for surgery or refuses surgery.

Precautions:

A well-fitted truss should prevent the descent of contents even if the patient stands up, stoops with his legs wide apart & coughs violently.

Complications:

1- Adhesions: If operation is required, it well be difficult.

2- Pressure causes local atrophy of muscles.

3- Badly fitting truss increases the risk of strangulation.

Contraindications:

1- Irreducible hernias.

2- Presence of other pathology that needs surgery e.g. varicocele.

3- Obese patients difficult to fit the truss properly.

II- Surgery:

"Correct the predisposing factors first"

A- Herniotomy: "Complete excision of the sac"

Indications:

Children and young adults with strong conjoint tendon & posterior wall of inguinal canal.

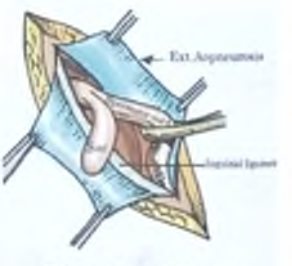
Steps:

1- Incision: 1 inch above and parallel to the medial 3/5 of the ing. ligament including skin & the two layers of superficial fascia (no deep fascia in the abdomen.).

2- Dissection of the cord: Open the external oblique aponeurosis down to the external ing. ring. The cord is identified, dissected & hooked.

3- Dissection of the sac: Open the cremastric muscle & fascia and internal spermatic fascia. The sac is identified and dissected up to the neck. It is transfixed & excised. The neck is identified by:

- It is the narrowest part.
- Appearance of extra peritoneal fat.
- Inferior epigastric vessels medial to it.



Dissection of the sac

4- Closure: in layers.

B- Herniorraphy: = Herniotomy + repair of the defect.

A) Repair of the posterior wall of the canal:

1) *Marcy repair*: just tightening (narrowing) of the internal ring.

2) *Lytle's repair*: plication of the fascia transversalis.

3) *Shouldice repair*: incision of fascia transversalis, then double breasting of it to reinforce the post. wall. It has a low recurrence.

Shouldice repair

B) Strengthening of the posterior wall of the canal:

1) *Bassini repair*: The most popular technique. The conjoint tendon is sutured to the inguinal ligament behind the cord.

Disadvantages:

- The sutures are under some tension.
- Interferes with the shutter mechanism.
- Fusion of muscle' to a ligament is weak.

Bassini repair

Tanner's slide: is a releasing incision in the rectus sheath to avoid tension on the repair.

2) *Halsted repair*: Bassini repair reinforced by suturing the two leaflets of external oblique aponeurosis behind cord.

Disadvantages:

- It interferes with the valvular mech. (loss of obliquity of the canal).
- The cord becomes superficial and liable to trauma.

Tanner's slide

3) *Bloodgood's repair:*

Reinforcement of the post. wall of the canal with a triangular flap from ant. rectus sheath that is turned laterally over its lateral border to be sutured to the inguinal lig .

Advantages:

a) It does not interfere with either shutter or valvular mechanism.

b) It is tension free.

4) Mc Vay (Cooper's lig) repair:

- Suturing the lower border of the transversus abdominis to Cooper's lig. medial to the femoral vessels, to the iliopubic tract above the vessels then back to Cooper's lig. lateral to the vessels.

- It is difficult and risky

Bloodgood's repair

C- Hernioplasty:

Reconstruction of the defect by using natural or synthetic patches.

1- Gallie's op. = Darning (رفو) with fascia lata.

The fascia lata is threaded over special (Gallie's) needle and darning starts from pubic tubercle to internal ring.

Darning

2- Mair's skin graft: Full thickness skin graft is de-epithelialized (to avoid development of dermoid cyst) & then placed over the post wall of the canal & sutured to both ing. lig & conjoined tendon.

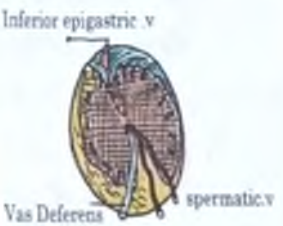
3) Lechtenestein repair: tension free hernioplasty

- The posterior wall of the canal is supported with a mesh.
- Prolene, Dacron, & Merseline mesh may be used.

Lechtenestein repair

4) Laparoscopic repair:

After laparoscopic excision of the sac, the back of the posterior wall of the canal is supported by a synthetic mesh clipped all-around and the peritoneum is closed from within.



Laparoscopic repair

OIH in females:

- The ovary is the usual content & liable for complications.
- Both the sac & round lig. of the uterus are excised & the inguinal canal is obliterated completely by closing the internal ring .

Sliding Hernia

Definition:

It is a hernia where a viscus slides to form a part of its wall.

Pathology:

- The commonest organ to slide is the urinary bladder.
- The sigmoid colon on the Lt. side and the caecum on the Rt. Side.

Graham's opertion

Clinical picture:

Sliding hernia is suspected if:

1- The hernia is not completely reducible.

2- The patient feels a desire of micturition on pressure on hernia.

Treatment:

Graham's method: a U shaped incision is done in the sac around the sliding organ. The gap in the sac is closed with sutures then the sliding organ is returned to the abdomen. The sac is ligated and removed.

Direct inguinal hernia

Definition:

It is a hernia through the weak Hasselbach's triangle.

Direct inguinal hernia

Aetiology: always acquired due to:-

1- Weakness of the conjoint tendon due to senility.

2- Paralysis of the conjoined tendon due to injury of the ilioinguinal nerve e.g. during previous appendectomy.

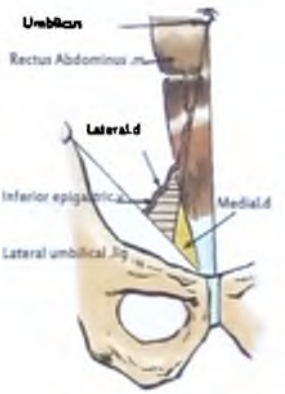
3- Precipitating factors include chronic cough, & straining e.g. SEP.

Pathology:

Site: Groin above the inguinal ligament.

Defect: Hasselbach's triangle .It is divided by the lat. umbilical ligament into two defects:

- *Lateral defect*: It is wide, so, the commoner lateral direct hernia is not liable for strangulation. The patient has a poor abdominal musculature.
- *Medial defect:* is narrow so, the less common medial (funicular or perivesical) direct hernia is liable to strangulation.



The two defects of the inguinal triangle

Sac: hemispherical with wide neck medial to the inf. epigast. art..

Contents: as before. Sliding urinary bladder is common.

Coverings: From outward inward.

1- Skin & subcutaneous tissue.
2- External oblique aponeurosis.
3- Conjoined tendon.
4- Fascia transversalis.
5- Extraperitoneal tissue.

Descent: Directly forward (very rarely, it passes through the external inguinal ring to pass with the cord (funicular type) to reach the scrotum.

Complications:

-They are rare due to wide neck, but the medial perivesical type is liable to strangulation.

-Sliding bladder is a common finding.

Incidence:

- It represents about 15% of inguinal hernia.
- It is always in males (never in females) & common in old age.
- It is bilateral in > 50% of cases.

Clinical picture:

Like OIH with the following differences:

Item	OIH	DIH
1- Incidence	Commoner in ♂ at any age	Only in ♂ usually old age
2- Side	More in Rt. side bilateral in30%	Bilateral in > 50y
3- Shape	Pyriform or tubular	Hemispherical
4- Direction of descent	Downward ,forward & medially	Directly forward
5- Descend into scrotum	May descend	Very rarely (funicular type)
6- Internal ring test	The contents don't descend	Contents descend
7- External ring test	The external ring is wide Impulses hit the tip of finger	The external ring is normal The impulses hit the finger side
8- Three fingers test	Impulses are perceived by the index which lies over int. ring	Impulses are perceived by the middle finger over the ing. Δ
9- Complications	Common	Rare (wide neck)
10- At operation	-The sac is inside the cord & lateral to the inf. epigastric art -Recurrence is uncommon	-The sac is outside the cord and med. to the inf. epigastric art. -Recurrence is common

Investigations:

Investigations for predisposing factors e.g. SEP. & chest infection.

Treatment:

"Treat the predisposing factors first".

I- Truss:

It has its place in direct hernia especially in small hernia with wide neck in old patients.

II- Operative treatment:

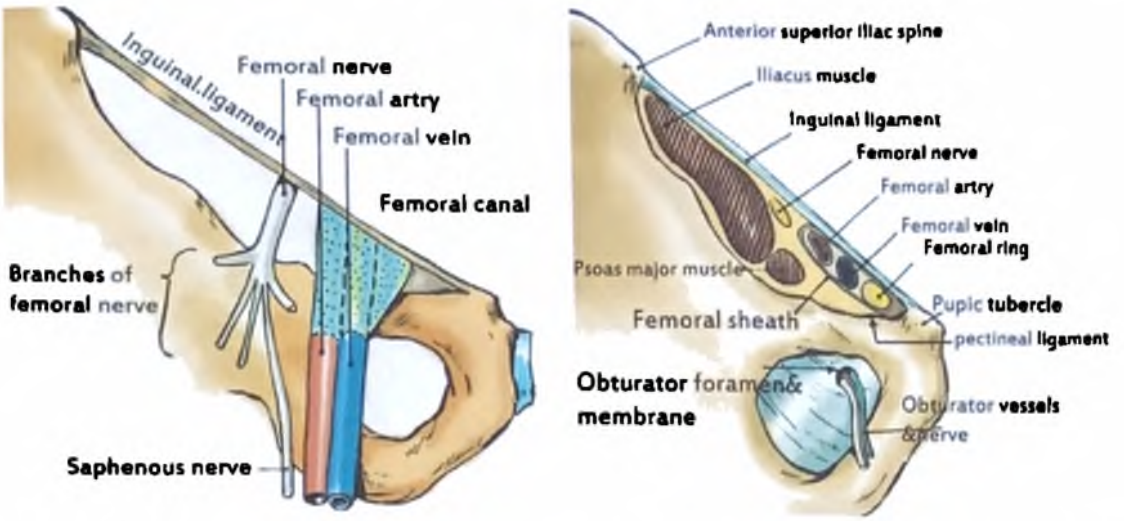
Indications:

Large hernia with narrow neck in a young patient.

Type:

- Herniorraphy: Like OIH but with following differences.
  - a- Bassini repair is not preferred as the conj. tendon is weak.
  - b- The sac is not opened but inverted by series of purse sutures in fascia transversalis.
  - c- Bloodgood repair is preferred because it is tension free.
- Hernioplasty: is the operation of choice.

Anatomy of the femoral canal



Femoral canal: is the medial compartment of the femoral sheath. It is not more than 1.5 cm long

Boundaries
- *Above* opens as femoral ring.
- *Below*: closed by fusion of the walls at the level of saphenous op.
- *Ant wall*: is formed by fascia transversalis.
- *Post. wall*: is formed by fascia iliaca .

Contents: Fat, lymphatics & LN. of Cloquet .

Function: It transmits lymphatics.

It allows expansion of the femoral vein during exercise.

Femoral ring:

Boundaries
- Anteriorly: Inguinal ligament (Poupart's lig).
- Posteriorly: Pectineal (Cooper's) lig.
- Medially: Lacunar lig. (the free sharp crescent margin) .
- Laterally: Femoral vein.

It is closed by a plug of fat called the femoral septum.

**Lacunar (Gimbernat's) ligament:*

- It is a triangular expansion from the medial part of the ing. ligament .Its apex lies at the pubic tubercle and it base is the free, sharp crescentic margin.

**Abnormal obturator artery:*

- In 30% of population, the obturator artery is absent and the pubic branch of epigastric artery descends behind the lacunar ligament and passes through the obturator foramen to replace the obturator artery.

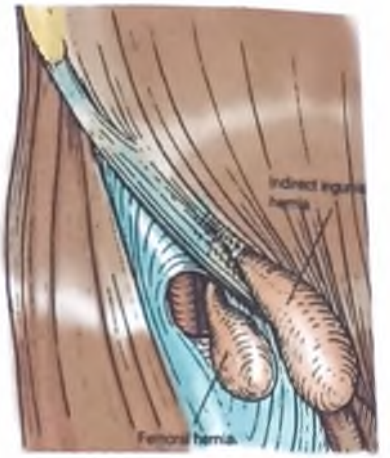
Femoral hernia

Definition:

External abdominal hernia through the femoral ring into the femoral canal.

Aetiology:

Always acquired due to increased intra-abdominal pressure.



Pathology:

Site: Groin below the inguinal ligament.

Defect: The femoral ring.

Sac: retort shape with narrow neck.

Contents: as before but Richter's hernia is common.

Coverings: from outward inward:

- Skin and superficial fascia.
- Cribriform fascia (covering the saphenous opening).
- Fascia transveralis (ant wall of femoral canal).
- Fat & lymphoid tissue (from the femoral septum).

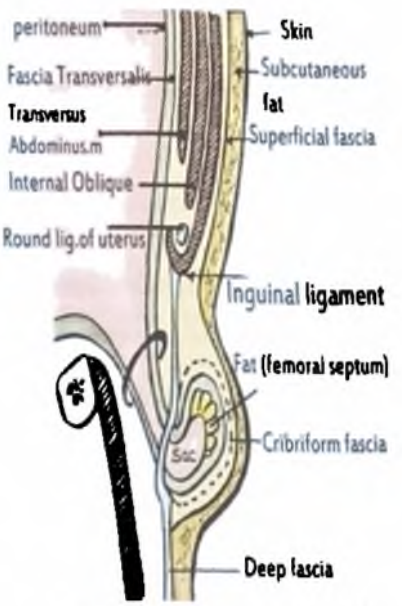
Descent: U shaped course.

Downward: through the femoral canal then

Forward: through the saphenous opening then

Upward: in the S.C. tissue may overlie the ing. lig.

- This is because of fusion of the superficial fascia of the abdomen to the deep fascia of the thigh at a line runs transversely from saphenous opening laterally.



Covering of femoral hernia

Complications:

- Irreducibility & strangulation are very common due to:
  a) Narrow neck with sharp edge of Gimbernat's lig.
  b) Sinuous course.

Incidence:

- It is the third commonest hernia (after inguinal & incisional hernias).
- It is more common in young adults (20-40y) females due to:
  - Wider femoral ring (wider pelvis, smaller vessels, & more slender iliopsoas).
  - Repeated pregnancy (intra-abdominal pressure - stretches pelvic ligaments)
  - More pelvic tilt brings the femoral canal with gravity.
- It is more common on the right (2:1) & bilateral in 20%.

Clinical picture:

- The patient presents with a swelling in the groin having the following characters:

1- Below and lateral to the pubic tubercle (but sometimes above inguinal ligament).

2- Reducible: Reduced downward then backward but practically impossible to be upward.

3- Shows impulse on cough.

-The patient frequently presents with complications e.g. strangulation.

Treatment: = **Surgery**

Truss is contraindicated due to:

1- Most femoral hernias are irreducible when first seen.

2- Strangulation is frequent.

3- Truss does not fit well in the upper thigh & the femoral ring lies obliquely.

Operations: Three approaches.

1- Low approach (Lockwood's or femoral approach):

- Incision 1cm below & parallel to inguinal ligament.
- The sac is dissected from below & transfixed as high as possible.
- Repair by suturing inguinal. Poupart's lig. to the pectineal lig.

Lockwood's approach

Advantages:

a- Rapid, easy & suitable for bad risk patients.

b- Inguinal canal is not opened.

Disadvantages:

a- The sac is not excised at the neck (high recurrence).

b- If strangulation is present.

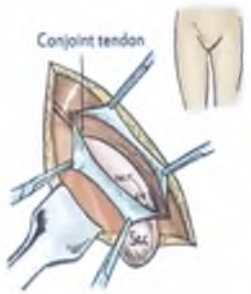
a- Blind cutting of Gimbernat's lig. may injure a possible abnormal obturator art.

b- If resection anastomosis of non-viable bowels is needed it cannot be done from below.

2- Lotheissen high (Inguinal approach):

Incision: Inguinal incision as in inguinal hernia.

- The inguinal canal is opened and fascia transversalis in the post wall of the canal is incised down to the public tubercle to expose the neck of the sac, which is dissected & excised.
- Repair is done by one of the following:-



Lotheissen approach

1- Suturing the Conjoined tendon to Cooper's lig (C to C).

2- Suturing Poupart's lig to Pectineal lig. (P to P).

3- Suturing Poupart's lig to the Conjoined tendon to the pectineal lig (P to C to P)

Advantages:

1- Excision of the sac at the neck (low recurrence)

2- If strangulated.

a- Injury of the abnormal obturator artery can be avoided.

b- Resection anastomosis can be performed through the same incision.

Disadvantages:

1- It needs experience.

2- It weakens the ing. canal (post wall).

3- In strangulated hernia, dissection of the sac from above is difficult.

3-Mc Evedy high approach

Incision: vertical incision over the sac and continued above over the ing. lig.

-Through the lower part of the incision the sac is dissected and through the upper part the dissection is completed to the neck and the sac is excised.

- Repair is by suturing the conjoined tendon to Cooper lig. (C to C)

Advantages:

Like Lotheissen's op. plus:

1-It does not weaken the post. wall of ing. Canal.

2-It is suitable for strangulated hernia.

Disadvantages:

1-Bad cosmetic incision.

2-infection leads to bad incisional hernia.

Mc Evedy high approach

Umbilical hernia

Types:

1- Congenital abdominal wall defect (exomphalos).

2- Congenital umbilical hernia.

3- Adult umbilical hernia.

I- Congenital abdominal wall defect

(Exomphalos)

It is congenital developmental defect of abdominal wall.

Aetiology:

Failure of all or portion of the midgut to return into the coelomic cavity during early fetal life

Pathology:

There are two types:

1- *Exomphalos minor* (hernia of umbilical cord or fetal omphalocele). The defect & sac are small .The umbilical cord is attached to the apex of sac.

2- *Exomphalos major* (embryonic omphalocele). The defect is wide and the sac is large with the cord attached to its inf. aspect.

Exomph. Major

Exomph. minor

Site: umbilical region.

Defect: umbilical abdominal wall defect (there is no umbilicus).

Sac: pouch of peritoneum.

Contents: Loop of small intestine or Meckel's diverticulum (exomphalos minor).
The whole midgut and portion of the liver (exomph. major).

Coverings: the thin sac is covered by-

- Thin outer layer of amniotic membrane.
- Thin middle layer of Warton's jelly.

Descent: directly forward.

Complications:

1- In exomph. minor, the contents may be included in ligature of the umbilical cord after birth .

2-In exomph. major :- a) rupture of the sac & peritonitis are great risks .
b) There are usually multiple cong. anomalies.

Treatment:

1- Exomphalos minor:

-Surgical correction is easy as the defect is small and the abdominal cavity is well developed.

-Surgical correction is performed by excision of the sac and closure of the defect in layers.

II- Exomphalos major:

(1) Surgical treatment:

In the first few hours of life, reduction of the contents into the small coelomic cavity is difficult & hazardous, so we try to cover the hernia with skin to avoid the risk of peritonitis. The skin & SC. fat are under-mined from the edge of hernia laterally with two relaxing incisions in the flanks. The skin flaps are approximated & sutured in the midline.

- Reconstruction of the abdominal wall & repair of hernia can be done months or years later when the coelomic cavity enlarges.

Surgical repair

(2) Expectant treatment:-

Indications:

1- Premature babies with large sac.

2- Poor general condition or major congenital anomalies.

Method:

- Painting the sac with mercurochrome 2%, which will form dry eschar over the sac that contracts gradually with skin growth over the amnion over weeks or months.

NB. Gastroschisis is an abdominal wall defect beside normal umbilicus and without coverings.

II- Congenital umbilical hernia

Aetiology:

-Yielding of the weak umbilical scar (may be due to neonatal omphalitis) in front of increased intra abdominal pressure (due to phimosis, coughing or crying).

Pathology:

Site: umbilical region.

Defect: incomplete closure of umbilical cicatrix.

Sac: Small, conical with wide neck.

Contents: Omentum or intestine.

Coverings: stretched umbilical scar, fascia of Piorier & extraperitoneal fat.

Descent: Directly forward.

Complications:

Obstruction or strangulation is rare.

Incidence:

Infants of both sexes, more common in males.

Clinical picture:

- Small reducible conical swelling at the umbilical scar that increases in size with crying, coughing or straining.
- It has a great tendency (>95%) to shrink & close spontaneously in the first year (s) of life (due to contraction of fascia of Piorier).

Treatment:

(Correct the predisposing factor first)

I- Expectant treatment:

Wait for spontaneous closure within 2 years with correction of the cause e.g. phimosis.

Indication:- Small hernia < 2 cm in diameter in young babies < 2 years.

Strapping: by a special umbilical truss that contains a hard cone that should be wedged over the defect to prevent descent of contents. It is claimed to hasten the closure, however, others think that it delays contraction of fascia of Piorier.

Surgical ttt (Herniorraphy)

Indications:

1- Large hernia (> 2 cm in diameter).

2- Failure of spontaneous closure for 2 years.

Incision

Through curved sub-umbilical incision, the hernial sac is dissected, excised at the neck and the defect in linea alba is closed with non-absorbable sutures.

III- Adult umbilical hernia
(Para-umbilical hernia)

Aetiology:

- It is due to defect in the linea alba above or below the umbilicus acted upon by increased IAP.

1- Defect: - Usually 90% above the umbilicus (supra umbilical hernia) .

- Occasionally below the umbilicus (infra umbilical hernia).
- Rarely in the umbilical scars (True umbilical hernia).

This is because the linea alba is wider and weaker above the umbilicus than below it & the umbilical scar is the strongest portion of linea alba.

2- Increased intra-abdominal pressure: e.g. repeated pregnancy, ascites.

Pathology:

Site: Umbilical region.

Defect: In the lines alba.

Sac: - Has a narrow neck and broad fundus.

- Multilocular due to:-

a- Adhesions between contents (omentum) & the fundus.

b- Fibrous septa derived from stretched linea alba.

Para-umbilical hernia

Contents: as before, but, omentum, small intestine & transverse colon are common.

Coverings: - Skin & superficial fascia.

- Stretched linea alba.

Descent: At first, directly forward then downward with sagging.

Complications:

1- Irreducibility, incarceration, strangulation are common due to:

a- narrow neck b- sharp edge of the defect c- adhesion & loculation.

2- Dyspepsia is common due to traction on omentum or mesocolon.

Incidence:

It is more common in ♀ (5 times), fatty, fertile (multiparous) between 30-50 years.

Clinical picture:

- Swelling: * In the umbilical region usually supraumbilical:-
  * showing Impulse on cough.
  * Reduced in backward direction, but, frequently irreducible.
- Dyspepsia is common due to traction on omentum & mesocolon.
- Dragging pain in huge hernia.
- Usually the patient presents with complications.

Huge PUH

Differential diagnosis:

I- From other causes of dyspepsia especially ch. cholecystitis (5F).

II-From epigastric hernia: PUH. lies within one inch from the umbilicus & causes distortion of the umbilicus.

Treatment:

Truss is not recommended due to-

1- Increased liability to complications.

2- The hernia is frequently incompletely reducible.

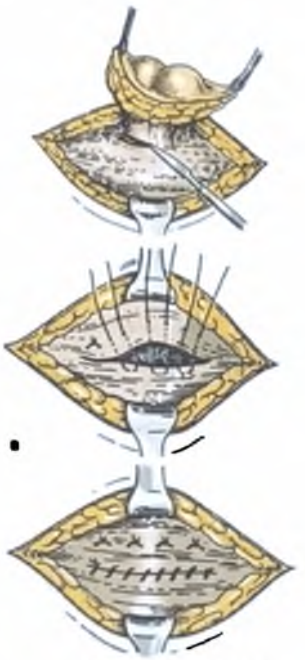
Surgery: is the only solution for cure.

I- Pre-operative preparation:

1- Reduction of weight is very beneficial.

2- Control of ascites & treatment of chest problems if present.

II- Operations:



Mayo's repair

1- Mayo's repair (transverse double breasting of the defect edges) was the most popular method used in the past but it proved high recurrence rate.

2- Hernioplasty with prolene mesh is the procedure of choice

3- Laparoscopic repair with fixation of mesh from inside is possible

N.B. We open the sac at the neck due to adhesions at the fundus & multiloculations.

Additional lipectomy & abdominopasty

In obese patient with pendulous abdomen, repair of the hernia can be completed with excision of a big ellipse of abdominal skin & fat (lipectomy).

Epigastric hernia

Aetiology: acquired.

Sudden straining tears the crisscross meshwork of linea alba.

Pathology:

Site: The epigastric area.

Defect: Small defect in linea alba at any site between xiphoid process to 1 inch above the umbilicus, usually midway between them and occasionally there are multiple defects.

Sac: Two types.

1- Fatty hernia of linea alba: only protrusion of extraperitoneal fat through the defect in linea alba without sac.

2- True epigastric hernia: in longstanding cases, a pouch of peritoneum is dragged to herniate through the defect.

Contents: The sac is either empty or contains portion of omentum.

Descent: directly forward.

Complications:

1- Irreducibility (very common). 2- Strangulation.

3- Dyspepsia (Traction on falciform lig. & omentum).

Incidence:

Common in young adult males especially manual workers.

Clinical picture:

1- Swelling in the epigastrium.

a- Soft, simulating lipoma.

b- Usually irreducible and may be tender.

c- Impulse on cough in true-epigastric hernia.

2- The patient may present with complications.

Fatty hernia of linea alba

Differential diagnosis:

1- From other swelling in the region e.g. lipoma.

2- From peptic ulcer & PUH.

Treatment: Surgery

- Transverse incision that is undermined for a suitable length rule out multiplicity

1-Small hernia with no sac → excision of the fat lobule with simple closure of the defect

2-Large hernia: may need hernioplasty.

Divarication of recti

Aetiology:

-Yielding of the linea alba by increased intra abdominal pressure due to repeated pregnancies or Hepato-splenomegaly (H.S.M).

Incidence:

It is common in three groups:

1- Babies 2- Young males with Hepato-splenomegaly 3- Elderly multipara.

Clinical picture:

-An oblong swelling appears in the midline above the umbilicus with straining .When the abdomen is relaxed, the fingers can be dipped into the gap between the recti.

Treatment:

I- In infants, the condition ceases spontaneously.

II- In the adult, abdominal belt is all what is required. Keel repair is rarely needed (see later).

Spigelian hernia

Definition: Hernia through the linea semilunaris (lat. border of rectus sheath) midway between the pubis and umbilicus.

Complications: incarceration & strangulation are very common.

Treatment: Surgery, *Herniorraphy is sufficient.*

Spigelian hernia

Incisional hernia

Definition:

Hernia at the site of previous abdominal incision e.g., operation.

Aetiology:

I- Pre operative causes (bad patient):-

1-Patients with poor musculature.

2-Patients with chronic straining conditions e.g. ch. Cough, ch. Constipation & bladder neck Constrictions.

3-Patients with debilitating diseases e.g. diabetes & malignancy.

II- Operative causes (bad technique):-

1- *Incision*: incisional hernias are common in:

a- vertical abd. incisions than transverse incision .

b- Muscle cutting incisions than muscle spitting or retracting incisions.

c- Incisions that cut nerves or bl. vessels to the muscles.

2- *Sepsis*: incisional hernias are common in septic operations e.g. operation for peritonitis.

3- *Difficult closure*: due to distended intestine or inadequate anaesthesia.

4- *Too tight sutures* (devitalization of tissues) or too loose sutures.

5- *Imperfect haemostasis* (haematoma→ infection) or *excessive undue haemostasis* → (ischaemia and infection).

III- Post operative causes (bad postoperative care):-

1- Wound infection.

2- Lifting heavy objects in the early post operative period.

3- Postoperative distension "Incisional hernia starts as asymptomatic partial burst".

Pathology:

Site: The site of previous incision.

Defect: The whole or part of the incision. The defect is common at the angles of the wound.

Sac: It is usually thin with variable size and irregular shape.

Contents: as before. and usually loops of small intestine.

Coverings: The sac always lies beneath the skin, which is stretched and may be atrophic.

Descent: Variable according to the site. Large hernias may sag downward.

Complications:

Irreducibility, incarceration and strangulation are common.

Incidence:

- It is the 2nd commonest hernia (after inguinal hernia).
- About 10% of abdominal operations are complicated by incisional. hernia.

Clinical picture:

- History of previous abdominal operation or incision.
- A swelling at the site of the scar showing impulse on cough (usually reducible).
- Cases may present with complications.

Investigations:

For detection of the cause e.g. anaemia, DM, & hypoprotienaemia.

Treatment:

I- Conservative treatment =

Palliative abdominal belt.

- Indicated only in patients with bad surgical risk: old age & immunodeficiency.
- The hernia should be reducible with wide defects.

II - Surgical:

A- Preoperative preparation: as in PUH.

B- Operations.

I- Herniorraphy

1) *Anatomical repair:*

- Indicated in small defects.
- After excision of the sac & the edge of the defect, the layers of the abd. wall are identified and sutured together separately.

2) *Cattle's five layers repair:* (historical)

.It was used in midline incisional hernia. The defect is then sutured in five layers

3) *Keel repair:*

Indications: 1- Wide defect.
2- Reducible hernia.
3- No loculation.

Keel repair

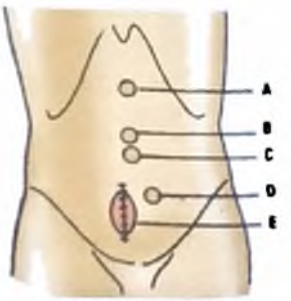
Principle:

The sac is not opened, but inverted by successive rows of inverting continuous sutures into the abdomen, thus the sac will finally project into the abdomen like the keel of a boat.

2- Hernioplasty (the only accepted method)

-Using skin graft or using synthetic mesh (proline, merseline .etc.)

N.B. Ventral hernias denote any hernia through the anterior abdominal wall rather than groin hernias. It includes the previous types e.g. epigastric (A), Paraumbilical & umbilical (B & C), Spigelian (D) & incisional hernias (E) and divarication of recti.



Ventral hernias

Burst abdomen

Definition:

- Dehiscence of abdominal wound with extraperitoneal prolapse of the viscera.

Aetiology:

The same causes of incisional hernia.

Pathology:

There are two types:

1- Complete burst: When all layers of the wound (including skin) give way, the bowels prolapse through the skin to the exterior.

2- Incomplete burst: When the deeper layers only give way with intact skin wound, the loops of intestine prolapse under the skin.

Complications:

1- Shock, dehydration & electrolyte imbalance.

2- Paralytic ileus & intestinal obstruction.

3- intestinal fistula.

4- Partial burst ends in incisional hernia.

Incidence:

About 1% of laparotomy incisions burst.

Clinical picture:

- *Serosanguineous discharge* from the wound is a warning sign.
- Usually between the 6^{th} to the 8^{th} day, the wound gives way and coils of intestines and omentum prolapse through the skin (complete burst). If the skin is intact over the loops of intestines, it is partial burst.

Treatment:

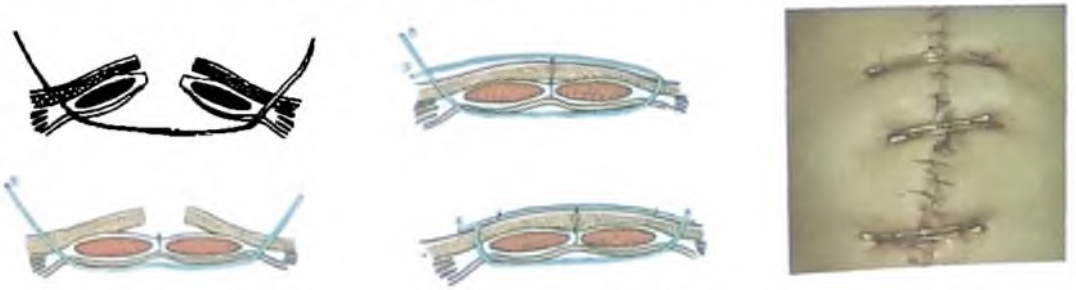
(A) Complete burst.

I- Resuscitation: awaiting for operation.

-Reassurance - cover the loops with sterile towels - I.V.fluid.

II- Operation: urgent.

- Washing the prolapsed loops with warm sterile saline and then replaced into the abdomen.
- All layers of the wound are approximated with *through & through sutures* of nylon passed through a plastic tube, so as, not to cut through the skin. This technique is modified by the author who uses plastic tubes in through & through sutures to approximate the two recti muscles supporting the wound & help healing without tension *(Lotfy's supporting tubes)*
- The abdominal wall is supported with strips of adhesive plaster



Lotfy's supporting tubes

(B) Partial burst.

It is treated by abdominal binder, delayed removal of sutures then, treated as incisional hernia.

Prognosis:

About 10% mortality rate.

Recurrent hernia

Aetiology:

1- Pre operative causes: (bad patient) as in incisional hernia.

2- Operative causes (bad technique):-

1-Failure to excise the sac completely (at the neck) e.g. sliding H.

2-Missing of another sac e.g. in Dual (pantaloon H.).

3-Under tension repair.

4, 5 like incisional hernia.

Incidence:

About 10% of hernias recur after primary repair within 1 year.

Treatment:

"Correct the cause first".

I- Conservative ttt: in patients with uncorrectable cause → truss.

II- Surgical ttt: of choice:-

1- Hernioplasty is usually needed.

2- In recurrent inguinal hernia in old patients, orchidectomy with excision of spermatic cord up to the int. ring and obliteration of the inguinal canal may be performed.

Rare external abdominal hernias

I- Lumber hernias:

1-*Inferior lumber hernia* through a defect bounded by iliac crest, latissmus dorsi & ext. oblique muscles.

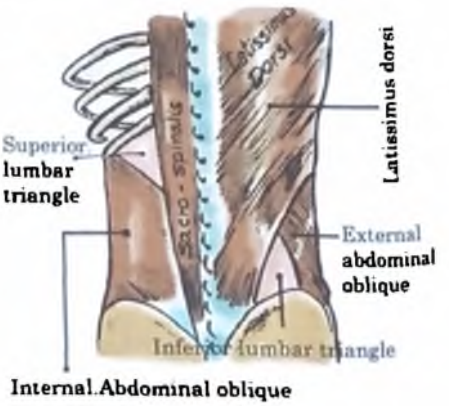
2-*Superior lumber hernia* through a defect bounded by the last rib, sacrospinalis & internal oblique muscles.

II- Obturator hernia: Through the obturator foramen.

III Gluteal hernia: Through the greater sciatic foramen.

IV - Sciatic hernia: Through the lesser sciatic foramen.

V – Perineal hernia: Through the peineal muscles



Sup. & Inf. lumber triangles

Diaphragmatic hernia

Types:

I- Congenital:

Due to persistence of pleuroperitoneal opening *(foramen of Bochdaleck)* or parasternal opening. *(Foramen of Morgagni).*

II- Acquired:

A- Traumatic diaphragmatic hernia:

-Major abdominal trauma with diaphragmatic tear. Herniation occurs immediately or years later.

-Traumatic hernia is common in Lt. side as the Rt. copula is supported by liver.

B- Hiatus hernia: through esophageal hiatus.

Diaphragmatic defects
1- Parasternal foramina
2- Oesophageal hiatus
3- Pleuroperitoneal foramina

Hiatus hernia

Types:

I- Sliding hiatus hernia (85%).

II- Paraoesophageal (Rolling) hiatus hernia.

III- Mixed hiatus hernia.

I- Sliding hiatus hernia

Definition:

-It is herniation (sliding) of the cardia and the upper stomach through the oesophageal hiatus into the thorax.

Aetiology:

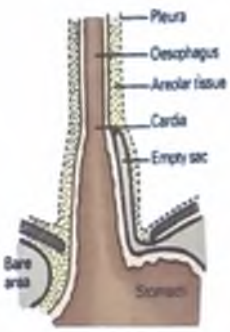
1- Muscle & ligaments weakness e.g. degeneration in elderly.

2- Increased intra-abdominal pressure: pregnancy, ascites, & S.E.P.

3- Obesity.

Pathology:

- The stomach draws a small sac (pouch of peritoneum) on the left side only (as on the right side there is the bare area of the stomach). This sac is small & empty.



Sliding hiatus hernia

- The cardia is drawn up into the thorax and becomes incompetent leading to reflux oesophagitis (all antireflux mechanisms are interfered with).

Complications:

Reflux oesophagitis with all its complications.

Incidence:

It is commoner in females over 40 years, fatty & fertile.

Clinical picture:

May be asymptomatic, or with manifestations of reflux oesophagitis.

Investigations:

1- Barium meal in Trendlenberg's position.

2- Oesophageal manometry & pH monitoring.

Differential diagnosis:

From reflux oesophagitis due to gall bladder disease.

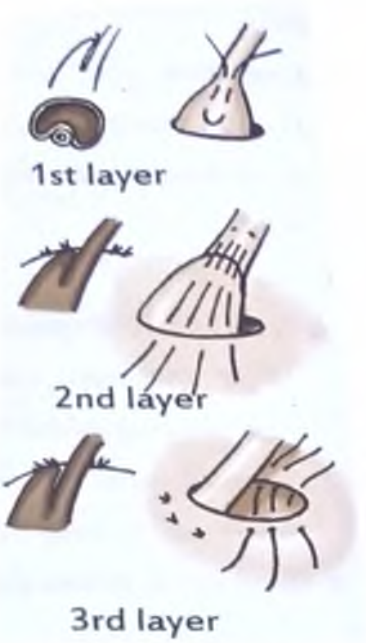
N.B. *Saint's triad: hiatus hernia, diverticulosis & gallstones.*

Treatment:

I- Asymptomatic cases →No treatment.

II- Mild cases →→ Conservative treatment, (see reflux).

III- Severe cases. → surgical treatment:



Belsy Mark IV op.

A- Transthoracic repair: *Belsy Mark IV operation.*

The repair is done in 3 layers:

First layer: Suturing the fundus to the anterior aspect of the oesophagus to restore *the angle of His* & warp the stomach 270° around the lower oesophagus.

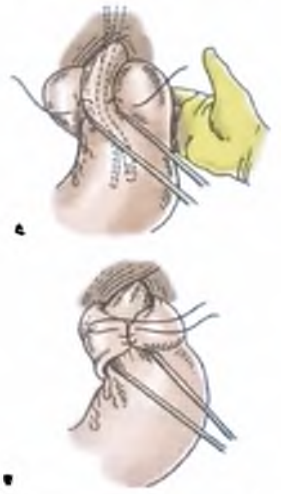
Second layer: Suturing the new oesophago-gastric junction to the undersurface of the diaphragm.

Third layer: Narrowing of the hiatus around the oesophagus.

B- Abdominal repairs.

1. Nissen's fundoplication:

-The fundus is wrapped 360° around the lower 5cm of the oesophagus to create a high-pressure jacket.



Nissen's fundoplication

Advantages:

1- It is an abdominal operation, easy and safer than Belsey.

2- It is more effective with no recurrence.

Disadvantages:

Gas bloat syndrome: failure of eructation, flatulence & dysphagia.

2-Toupet's fundoplication:

Wrapping the fundus 270° only around the lower oesophagus with less postoperative dysphagia and gas bloat syndrome.

3-*Hill's procedure*: (posterior gastropexy): Suturing the cardia to the Lt. crus and median arcuate ligament with narrowing of the hiatus.

Hill's gastropexy

4- *Angelchick prosthesis*: after reduction, a plastic ring is fixed around the cardia to prevent herniation. It may cause ulcer or stricture.

5-*Collis gastroplasty*: In cases with short oesophagus, a gastric tube along the lesser curvature is fashioned to lengthen the oesophagus with narrowing of the hiatus.

Collis gastroplasty

II- Paraoesophageal (Rolling) hernia (10%)

Definition:

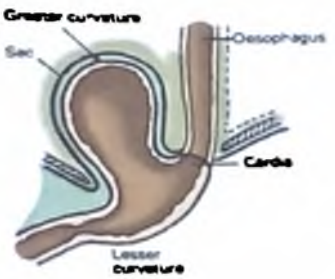
- Herniation of the stomach through the oesophageal hiatus beside undisplaced gastro-oesophageal junction within a peritoneal sac.

Pathology:

- Normally placed cardia → no reflux oesophagitis.
- Pressure on mediastinal structures → pressure symptoms.

Clinical picture:

- Cardio-respiratory symptoms: palpitation and dyspnea.
- Dysphagia (pressure on oesophagus).



Paraoesophageal hernia

Investigations:

Barium meal & endoscopy.

Treatment:

Belsey Mark IV operation.

III- Mixed hiatus hernia

Definition: Both sliding & rolling varieties coincide.

Clinical picture: Combination of both reflux & pressure symptom.

Treatment: Belsey Mark IV or Nissen's fundoplication.

Mixed hiatus hernia

Complicated hernias

1- Irreducible hernia

Definition:

Failure of reduction of the contents with no evidence of other complications.

Aetiology:

1- Adhesions between the contents and the sac & among the contents.

2- Oedema of the contents. or narrowing of the neck by adhesions.

Clinical picture:

- The contents fail to return to the abdomen but:
  - *There is still impulse on cough.
  - *The hernia is not tense or tender.

Treatment:

- Early operation in the earliest convenient time to avoid strangulation & obstruction.
- The sac is opened. adhesions are cut, contents are reduced then repair is done.

II- Inflamed hernia

Definition:

Inflammation of the contents.

Aetiology:

1- External pressure by ill-fitted truss.

2- Internal inflammation of the contents e.g. appendicitis or salpingitis.

Clinical picture:

- General constitutional manifestations such as fever, tachycardia, & malaise.
- Local signs of inflammation: pain, tenderness. redness, oedema but the hernia is not tense.
- Impulse on cough can be elicited.

Treatment:

- Expectant: (preoperative) antibiotics, & antiphlogestic poultice.
- Surgical ttt: Dealing with the contents during operation e.g. appendicectomy.

III- Obstructed hernia

Definition:

An irreducible hernia containing loop of intestines whose lumen is occluded but without interference to its blood supply.

Clinical picture:

General: General manifestations of int. obstruction i.e. colic, constipation, distension, & vomiting.

Local: -The hernia is irreducible and somewhat tender but:

* Impulse in cough may be elicited with difficulty.
* It is not tense but indentible (incarcerated hernia.)

Treatment:

I- Conservative treatment: repeated oily enemata may relieve obstruction.

II- Surgical treatment: Obstruction is relieved by division of adhesions or the narrow neck.

Strangulated hernia

Definition:

An irreducible hernia with interference to the blood supply of its contents.

Actiology:

1- Narrow neck, adhesions, sharp edge of the defect.

2- Sudden descent of new contents.

Pathology:

Sac: It loses its luster and becomes distended with fluid, which is black, offensive, and with high bacterial content (migrating from the bowel).

Contents: See strangulated intestinal obstruction.

Coverings: Inflamed (edematous & congested).

Incidence:

- The most liable hernia to strangulate is the femoral hernia (25%) and the PUH (15%) However the commonest hernia seen strangulated is the ing. hernia being the commonest one.

Clinical picture:

General: Signs of strangulated intestinal obstruction:-

Pain (colicky & dull aching), vomiting constipation and distension.

These manifestations are absent or delayed in:

1-strangulated omentum 2-strangulated Littre's hernia 3-strangulated Richter's hernia

Local: Cardinal signs of strangulation:-

- The hernia is irreducible.
- No impulse on cough.
- It is tense & tender.

Differential Diagnosis

From irreducible, inflamed & obstructed hernia:

Item	Irreducible	Inflamed	Obstructed	Strangulated
- Intestinal obstruction	-	-	+	+
- Reducibility	-	-	-	-
- Impulse on cough	+	+	+(difficult to elicit)	-
- Tenderness	-	++	+	++
- Tense	-	-	-	+

Treatment:

A- Resuscitation:

- I.V. fluids. - Antibiotics. - N/G suction.

B- Taxis (obsolete):

Forcible reduction of hernia within 6 hours of the onset.

Complications:

1- Rupture of the sac. 2- Rupture of the strangulated loop

3- Reduction of gangrenous loop. 4- Reduction en block (sac & contents)

C- Surgery: Urgent operation.

1- Wide incision.

- Inguinoscrotal in inguinal hernia.
- Modified Lotheissen or inverted L shaped incision in femoral hernia.

2- Dissect the sac and open it at the fundus to evacuate the toxic fluid. In PUH, the sac is opened also at the neck.

3- Division of adhesions and the constricting ring and the contents are pulled out and inspected (Maydle's hernia should be considered).

4- The contents are dealt with accordingly.

- Omentum: resection. whether viable or not.
- Intestines
  - Viable → reduction
  - Doubtful: → O2 ventilation, and observation for signs of viability.
  - Non-viable: → resection anastomosis. If small intestine or right colon Exteriorization resection in Lt. colon.

5-Herniorraphy is done but hernioplasty is better avoided as the mesh usually gets infected.

6-Closure with drains.

THE OESOPHAGUS

Although the oesophagus is mainly a thoracic organ, yet it is better discussed with abdominal organs because most of its surgery is through the abdomen in association with stomach.

Embryology:

The oesophagus develops together with the trachea from the cranial part of the endodermal tube (the future foregut). A longitudinal septum appears and begins to separate the trachea ventrally and the oesophagus dorsally but, still communicating with each others cranially at the pharynx.

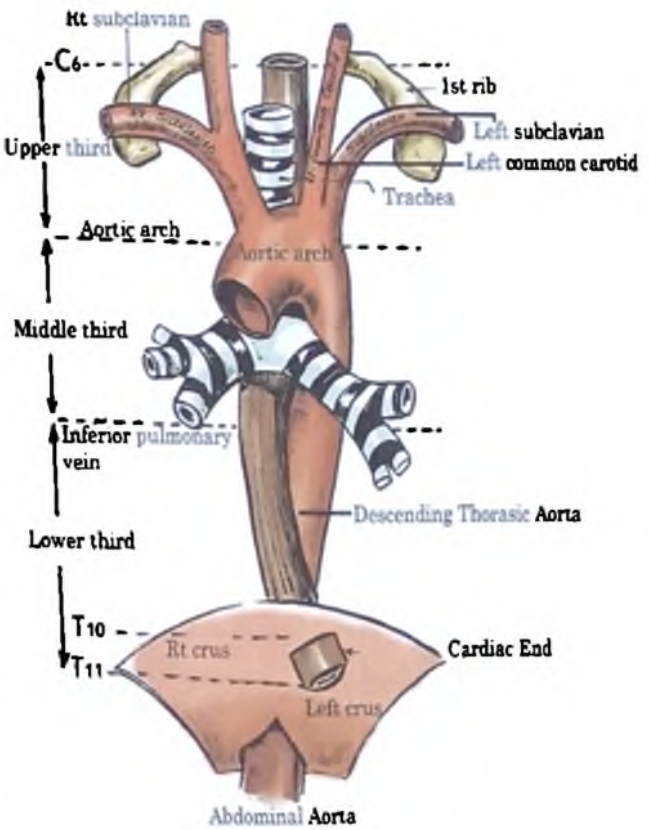
ANATOMY:

It is 25 cm long muscular tube connecting the hypopharynx to the cardia of the stomach.

Begins: at the lower border of the hypopharynx behind the cricoid cartilage at the level of sixth cervical vertebra (C6)

Course: It descends in front of the vertebral column in the neck, superior then posterior mediastinum behind the trachea. To the right, then in front then to the left of the descending thoracic aorta. It pierces the diaphragm at the level of (T_{10}) with both vagi to enter the abdominal cavity behind the Lt. lobe of the liver.

Ends: At the cardiac end of the stomach at the level of (T_{11}).



Blood supply:

1) **Arterial** :

-*Cervical part:* by branches from the inf. thyroid artery.

-*Thoracic part*: by branches from the descending thoracic aorta.

-*Abdominal part*: by branches from the Lt. gastric artery.

2) **Venous**:

- *Cervical part*: into the inf. thyroid vein.
- *Thoracic part*: into the azygous and hemiazygous veins (systemic).
- *Abdominal part*: into the Lt. gastric vein (portal).

Lymphatic drainage:

- *Cervical part*: drains into deep cervical lymph nodes (supraclavicular lymph nodes).
- *Thoracic part*: drains into the paraoesophageal. & tracheo-bronchial LNs
- *Abdominal part*: drains into the Lt. gastric lymph nodes → coeliac LNs

Nerve supply:

- *Cervical part*: Parasympathetic comes from the recurrent laryngeal nerves (vagi).
  Sympathetic supply comes around the inferior thyroid artery.
- *Thoracic part*: Parasympathetic from oesophageal plexus.
  Sympathetic from greater splanchnic nerve.
- *Abdominal part*: by the anterior and posterior gastric nerves (the Vagi).

Applied anatomy

- The oesophagus has five constrictions:-
  1. Cricopharyngeal constriction at its beginning.
  2. Aortic constriction, where it is crossed by the aortic arch.
  3. Pulmonary constriction, where it is crossed by the Lt. main bronchus.
  4. Diaphragmatic constriction at the oesophageal hiatus.
  5. Sphincteric constriction at its end.

  These sites are:
  - -Sites of arrest of swallowed foreign bodies.
  - -Most exposed parts to mechanical and chemical trauma (corrosives).
  - -Common sites for malignancy.
- The oesophagus is surgically divided into three parts:
  1. *Upper third* from its beginning to the aortic arch (cervical incision)
  2. *Middle third* from its aortic arch to the level of inf. pulmonary vein (thoracotomy incision).
  3. *Lower third* from the inf. pulmonary vein to the cardiac (end abdominal incision).
- The lower end of the oesophagus is one of the most important sites of Porto-systemic anastomosis, where the left gastric vein (portal) anastomoses with tributaries of the azygous and hemiazygous veins (systemic). In portal hypertension, these anastomotic veins dilate forming what is called oesophageal varices.
- Surgical anastomosis in the oesophagus carries high risk of leakage because:
  1. The oesophagus is devoid of serous covering except its abdominal part.
  2. The muscular layer is thin and weak poorly holding the sutures.
  3. The oesophagus is poor in blood supply.

I-Congenital anomalies

(1) Congenital atresia or stenosis with or without fistula

Aetiology:

Failure of complete canalization of a segment in the oesophagus with or without failure of complete septum formation between oesophagus and trachea.

Pathology

Types:

Type I: atresia with fistula to the lower pouch 85%.

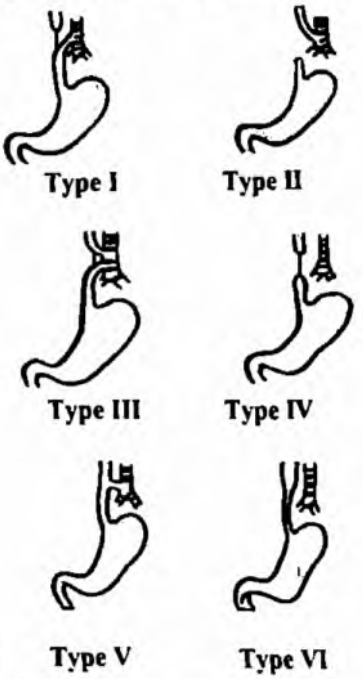
Type II: atresia with fistula to the upper pouch 2%.

Type III: atresia with fistula to both pouches 1%.

Type IV: atresia without fistula 12%.

Type V: fistula without atresia (H type anomaly).

Type VI: stenois without fistula.



Clinical picture:

Manifested at birth with:

1- Drolling of saliva from the mouth & inability to swallow.

2- Regurgitation of food with attacks of choking and cyanosis

3- Abdominal distension and flatulence in cases of fistula to the lower segment.

4- Failure of a catheter to pass to the stomach and is arrested 10cm from the incisor teeth.

NB. It may be a part of VACTERL syndrome (Vertebral, Anorectal, Cardiac, Tracheo-Esophageal, Renal and Limb anomalies)

Investigations:

1- Plain X-ray abdomen in erect position. If it shows gas in the stomach, this denotes a fistula to the lower pouch. Gasless abdomen means blind lower pouch.

2- Injection of small amount of lipidol into the catheter detects fistula to the upper pouch.

Treatment:

-Treatment is surgical and should be done as early as possible.

-If the gap between the two pouches is small, the fistula is excised, if present, and the two pouches are anastomosed together around a catheter.

-If the gap is large, the upper pouch is opened up in the neck (oesophagostomy) to drain saliva. The fistula is excised, and gastrostomy is done for feeding until a colon bypass is carried out later.

(2) Congenital diverticula:

Commonly in the midthoracic part

Oesophageal diverticula

1- Zenker's diverticulum (pharyngeal pouch): See before.

2- Midthoracic diverticula:

-Congenital diverticula.

-Pulsion diverticula, which occur due to increased intraluminal pressure as in case of motility disorders, e.g. diffuse oesophageal spasm.

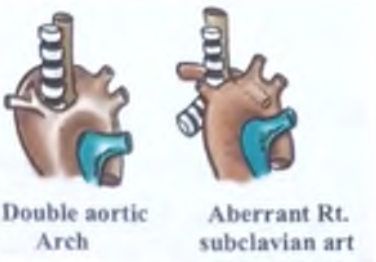
-Traction diverticula due to nearby fibrosis e.g. mediastinal tuberculous LNs.

3- Epiphrenic diverticulum: It is usually of the pulsion type

3) Dysphagia lusoria (الخادعة)

Aetiology:

Compression of the oesophagus by congenitally abnormal vessels e.g. double aortic arch encircling the oesophagus and trachea or aberrant Rt. subclavian artery arising from descending thoracic aorta.



Double aortic Arch | Aberrant Rt. subclavian art

Clinical picture

Although congenital in nature, yet the condition is often discovered or manifested after the age of 50 years where atherosclerosis develops and the rigid arterial wall compresses the oesophagus causing dysphagia (DD. cancer oesophagus).

Treatment:

Division of the constricting vessel if possible or vascular reconstruction

II- Trauma

Corrosive Injury

Definition

Chemical burn of the esophageal mucosa by strong acids (most injurious), alkalies or others.

Pathology:

- It is usually a deep burn, which commonly leads to firm stricture.

Complications:

1- Stricture 2- fistula with trachea or pleura 3- carcinoma

Incidence

Common in children (accidental) or adult (suicidal).

Clinical picture:

- History of ingestion of a caustic material is usually positive.
- Pain, dysphagia and may be dyspnea and stridor.

Investigations:

Early, investigations are not needed. Later, barium swallow and endoscopy are done to evaluate the degree of stricture

Treatment:

(1) Immediate treatment:

- Dilution of corrosive by water or egg white starch solution.
- Chemical neutralization is contraindicated as it produces gas & heat and precipitates ruptu
- Gastric lavage is contraindicated.
- Urgent tracheostomy is indicated in case of stridor.
- Feeding gastrostomy is sometimes indicated.
- Corticosteroids should be given for 6 weeks to minimize oedema and fibrosis.
- Antibiotics to prevent respiratory infection or mediastinitis.
- No attempt should be made for oesophagoscopy or dilatation during the 1st week, as there is high risk of perforation.

(2) Subsequent treatment:

a) After 1 week, barium swallow is performed. If it shows stricture, an attempt should be done to dilate the stricture with bougie through oesophagoscopy, followed by regular dilatation.

b) *Surgical treatment:* (in about 40% of cases).

Indicated in:

1-Tight stricture. 2 –Fistula with trachea or pleura 3- Failure of dilatation.

Procedures:

- Preliminary gastrostomy for feeding and improving the general condition of the patient.
- Colon bypass (usually retrosternal to allow normal growth).

Traumatic perforation

Causes: penetrating injuries, endoscopy, dilatation of strictures &foreign bodies.

Pathology: leak leads to mediastinitis, empyema, cervical abscess & peritonitis

Clinical picture: pain, fever, toxemia, surgical emphysema, mediastinitis or peritonitis.

Investigations: plain x ray .... Emphysema

CT with contrast to detect site of leak

Treatment: antibiotics, drainage, and surgical repair as early as possible.

III-Inflammation

Reflux oesophagitis

Definition:

Chronic oesophagitis due to regurgitation of gastric acid into the oesophagus.

Aetiology:

1) Primary reflux due to incompetence of the lower oesophageal sphincter mostly due to sliding hiatus hernia (H.H).
2) Secondary reflux: secondary to delayed emptying of the stomach due to:

a-Pyloric stenois b-Pyloric spasm. : DU, gallstone, ch. Appendicitis.

***N.B.** Antireflux mechanisms:*

1) Valvular mechanism of oesophagogastric angle *(angle of His)*.
2) Pinch-cock like action of the Rt. crus of the diaphragm.
3) Rosette like arrangement of the cardiac gastric mucosa.
4) The lower oesophagus is intra-abdominal and squeezed by the intra-abdominal pressure.
5) The sphincteric function of the circular muscle fibres around the lower end.

Pathology:

Grading: *Grade 1*: Reddness of the mucosa without ulcerations.

Grade 2: Linear ulcerations that bleed easily.

Grade 3: Coalesced ulcerations that leave islands of epithelium.

Grade: 4: Stricture

Complications:

1) Peptic ulcer in the lower oesophagus.
2) Bleeding.
3) Acquired short oesophagus.
4) Oesophageal stricture.
5) Barrett's oesophagus.
6) Malignancy.

Incidence:

- Common in middle age male.

Clinical picture:

- Heartburn: burning pain deep to the sternum, which is aggravated by stooping or lying down, but relieved by sitting and intake of alkalies.
- Water brash is due to reflex salivation in response to irritation of the mucosa by gastric acid
- The patient may present with atypical presentation e.g. angina pectoris, bronchial asthma, chocking attack or dental caries.
- The patient may present with one or more of the complications.

Investigations:

1-*Barium meal* in Trendlenberg's position may reveal:

-The reflux - the causative H.H. -stricture.

2- *Oesophagoscopy* may show -mucosal ulcer, leukoplakia or the reflux.

3- *Oesophageal pH monitoring* confirms the diagnosis.

Treatment:

A- Conservative ttt: successful in about 80% of cases

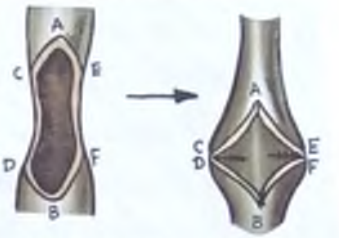
1-Elevation of the head of the bed.

2-Avoid stooping, heavy work, irritant food, and ulcerogenic drugs.

3-Regulation of diet (six small soft spiceless slowly masticated meals in standing position).

4-Drugs: - antacids, H_2 receptors antagonists and omeperazole for symptomatic relief.

B) Surgical treatment:



Thal's patch operation

Indications:

1- Resistance to or recurrence after vigorous medical ttt.

2- Development of complications e.g. stricture

Procedures:

1. Anti reflux operations (see ttt of hiatus hernia).

2- Treatment of complications e.g. stricture →*Thal's patch operation* (Longitudinal incision that is closed transversly. that may be supported with patch from the wall of the stomach)

IV-Neuromuscular disorders

1- Cardiac achalasia (cardiospasm)

Cardiac achalasia (mega-oesophagus).

Definition.

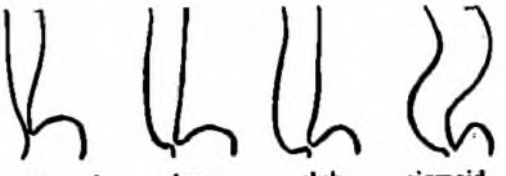
Failure of relaxation of the cardiac sphincter in response to swallowing leading to progressive oesophageal dilatation (mega-oesophagus).

Aetiology:

It is a neuromuscular incoordination due to loss of ganglion cells in Auerbach's plexus. Psychic emotions, Vit B deficiency and trypanosomaisis cruzi (Chagas disease) were incriminated.

Pathology:

1-Weak or absent peristalsis in the body and failure of relaxation of the lower oesophageal sphincter to relax (*functional obstruction*).



2- Progressive oesophageal dilatation: (funnel→ cigar → club → sigmoid shaped oesophagus).

Complications:

1- Weight loss 2- inhalation pneumonia 3-carcinoma

Incidence

Common in young adults with emotional lability. No sex predeliction.

Clinical picture

1- Dysphagia which is at first intermittent, of long duration, more to fluid than to solids because solid food initiates stronger peristalsis and opens the cardia by their weight.

2- *Regurgitation* of undigested food.

3-*Retrosternal fullness* and discomfort.

4-The patient may present by one of its complications

Investigations

I- Barium swallow shows:

1) Air fluid level behind the heart with absence of gastric air bubble in plain X-ray due to the presence of constant water seal by the stagnant fluid preventing air to pass to the stomach.
2) Dilatation of the oesophagus giving cigar, club, funnel or sigmoid shape appearance.
3) Smooth tapering of the lower end of the esophagus, which ends below the level of the diaphragm giving what, is called *hen's or parrot peak appearance*.

II- Oesophagoscopy: may reveal:

- Dilated proximal oesophagus.
- Fluid level of the stagnant food (most diagnostic).
- Eccentric pinpoint cardia.

III- Oesophageal motility studies: Manometry shows:

- Weak feeble contractions in the body of the oesophagus.
- The pressure in the lower end does not drop during swallowing.

Achalasia of the cardia

Differential diagnosis

From other causes of dysphagia especially carcinoma. (see later).

Treatment:

I) Conservative ttt: gives temporary relief in the form of:

- Psychotherapy in psychic cases
- Octyl nitrite (smooth muscle relaxant) or Nifedipine (Ca^{++} channel blocker) before meals.

II) Minimal invasive therapy: It includes

1- Endoscopic hydrostatic dilatation: Dilatation up to 5cm diameter to rupture the sphincter by "*Plummer's hydrostatic bag*". It is dangerous and may lead to perforation.

2- *Endoscopic injection of botulinum toxin (Botox) into the sphincter:* It may paralyze the sphincter up to 6 months and may bring permanent relief.

III) Surgical ttt:

Heller's operation (oesophago-cardiomyotomy): single anterior longitudinal incision is done through the longitudinal & circular muscle fibers of the lower oesophagus until the mucosa of the oesophagus bulges out. Some surgeons prefer to add an antireflux procedure although, short cardiomyotomy is usually sufficient and usually does not lead to reflux oesophagitis. It can be done laparoscopy

2) Diffuse oesophageal spasm

Cork-screw oesophagus

A motility disorder characterized by non-peristaltic contractions giving "*Cork screw appearance*" in barium swallow.

- It presents with dysphagia with chest pain.

Treatment: long oesophagomyotomy up to the aortic arch

V- Neoplasia

A: Benign tumours: the commonest is leiomyoma, also lipoma, adenoma, and fibroma.

B: Malignant tumour: carcinoma.

Cancer oesophagus

Aetiology:

I) predisposing factors:

Chronic irritation: 5Ss (Sepsis, Syphilis, Smoking, Spices, and Spirits).

II) Premalignant conditions:

1- Achalasia
2- reflux oesophagitis and Barrett's oesophagus
2- leukoplakia
4-Plummer Vinson syndrome.

NB. *Plummer Vinson syndrome* is a rare disease of young females and consists of

- Anaemia (iron def.)
- Spasm of cricopharyngus m
- Atrophic glossitis
- Spooning of nails
- Achlorohydria
- Splenomegaly.

Sites

Pathology:

Sites: common in the anatomical narrowing.

Upper 1/3: 20% middle 1/3: 30% lower 1/3: 50%

Macroscopically (MAC):

1- Annular infiltrative type: more in the lower 1/3. :

2-*Ulcerative type*: huge ulcer with everted edge, necrotic floor and indurated base, which extends beyond the ulcer margin.

3- *Fungating type*: a cauliflower mass.

Microscopically (MIC):

1- Squamous cell carcinoma (95%) usually poor differentiated (no cell nest or keratin).

2- Adenocarcinoma (5%) from either heterotropic gastric mucosa or on top of Barrett's epith.

Spread:

1) Direct spread :

a*) Intrinsic* in the wall longitudinally 1 inch beyond the visible margin and deeply .

b) *Extrinsic:* into the surrounding structures: e.g. trachea, pleura, aorta, Lt. lobe of the liver

2) *Lymphatic spread*: see anatomy.

3) *Blood spread* the upper 1/3 → lung & the lower 2/3 → liver.

Complications (causes of death):

1) Malnutrition, cachexia, dehydration.

2) Inhalation pneumonia.

3) Invasion of the nearby structures.

-*Pleural cavity* → empyema

-*pericardium* → purulent pericarditis.

-Tracheobroncial tree → pneumonia

-aorta → fatal haemorrhage.

-Rec. laryngeal n. → hoarseness of voice

-phrenic n. → hiccough & diaphragm paralysis.

-mediastinum→.fatal mediastinitis.

4) Distant metastasis.

Incidence:

- Common in males (80%) above the age of 40 years but may affect any age.

Clinical picture

1-Dysphagia (malignant) which is:

-Progressive - of short duration -more to solids than fluids (organic)

2- Regurgitation: less than achalasia due to short duration and anorexia.

3-*Retrosternal discomfort* during swallowing is an early symptoms but pain is usually late.

4-*The patient may present with one of the above complications.*

Investigations:

Rat tail appearance

I-Barium swallow: - shows.

1) Annular carcinoma: irregular stricture which is relatively long extending above the diaphragm giving (rat tail appearance), shouldering with no or slight proximal dilatation due to short duration & malignant anorexia.

2) Ulcerative & fungating carcinomas: They give irregular filling defect.

II- Oesophagoscopy and biopsy: the most important investigation.

III- Endoscopic U/S: It shows the degree of wall infiltration & perioesophageal LNs. spread.

III- C.T. scan and abd. US. : to assess operability .

Differential diagnosis:

From other causes of dysphagia (see later).

Dysphagia in a male above 50 years, more than 2 weeks should be considered cancer oesophagus until prove otherwise.

It is differentiated from achalasia by the following items:

Items	Achalasia	Cancer oesophagus
Type of patient	Young adult female	Middle age or old male
Course	Intermittent	Progressive
Duration	Long	Short
Dysphagia	More to fluids	More to solid
Regurgitation	Common	Less common
General condition	Relatively good. Wasting is late	Bad and cachectic
Barium swallow	Hen's peak appearance.	Rat tail appearance
Oesophagoscopy	diagnostic	diagnostic

Treatment

A) Inoperable cases :

Unfortunately, most cases are inoperable.

Criteria of inoperability are:

General:

1- Cases unfit for surgery.

2- Presence of distant metastasis

Local:

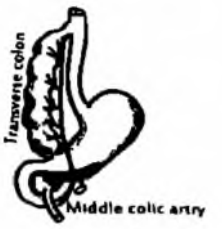
1-Irresectable tumour

2- distant lymphatic spread.

-Palliative radiotherapy: to prevent or postpone complications.

-Palliative surgery: to bypass obstruction as follows:

1) Gastrostomy: no longer performed, as
   * It does not prevent inhalation pneumonia by accumulated saliva.
   * It has bad psychological impact
   * Lack of food tasting.
2) Colon bypass: Antesternal (subcutaneous). Either the right or the transverse colon is used
3) Oesophagojejunostomy: Roux en Y to avoid biliary oesophagitis
4) Gastric tube to bypass lower one-third obstruction.
5) Insertion of Souttar tube through the growth after Laser tunneling of the tumour. Now, endoscopic dilatation and stenting is the best



Colon bypass

Oesophagojejunostomy

Gastric tube

Souttar tube

B) Operable cases:

I- Endoluminal treatment:

- For very early superficial disease and dysplastic mucosa
- Radiofrequency ablation (RFA), laser, argon plasma coagulation.

II_ Surgery

Preoperative preparation:

1-Correction of dehydration or electrolytes deficit if prese

2- Treatment of any chest infection

Surgical rule

- Regardless of the site of the tumor, the limits of excision are:-
  - *The proximal cut is 2 inches above the tumour.
  - *The distal cut should be in the stomach to pull it up
- Pyloroplasty may be added as the vagi are cut during the procedure

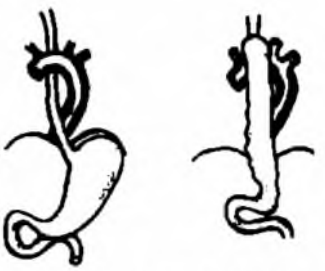
I- Carcinoma of the upper 1/3:

Total oesophagectomy = Mc keown operation.

Aim: to remove the whole oesophagus below the tumour and 5 cm above it with gastric pull up to be anastomosed with the oesophagus in the neck.

Incision: 3 incisions:

1) Midline laparotomy to assess operability & to mobilize the stomach.
2) Rt. 5th thoracotomy to mobilize the thoracic oesophagus to avoid the aortic arch on the Lt. side and to ligate the azygos vein on the right side


Mc keown operation

3) Rt. oblique cervical incision along the anterior border of the Rt. sternomastoid to expose the cervical oesophagus (Why on the Rt.?) to avoid injury to the thoracic duct on the Lt. side

NB. Recently, transhiatal resection of the oesophagus (oesophagectomty without thoracotomy) is preferred, where dissection of the oesophagus can be done blindly through the oesophageal hiatus and the stomach can be pulled up through the cervical incision without the need for the thoracotomy and hence, lowers morbidity and mortality.

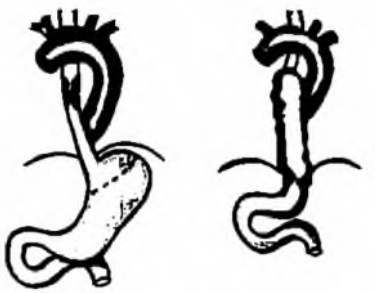
II- Carcinoma of the middle 1/3:

Partial oesophagogastrectomy = Ivor Lewis operation.

Aim: to remove the upper 1/5 of the stomach together with the whole oesophagus below tumour and 5 cm above it with gastric pull up to be anastomosed with oesophagus in the thorax.

Incisions: 2 incisions:

1) Midline laparotomy
2) Rt. 5th thoracotomy



Ivor Lewis operation

N.B: Some surgeons prefer to perform total oesophagectomy even in the middle third so that the anastomosis is done in the neck not the thorax, thus, if leakage occurs from the anastomosis, it will be in the neck and not in the mediastinum or pleura.

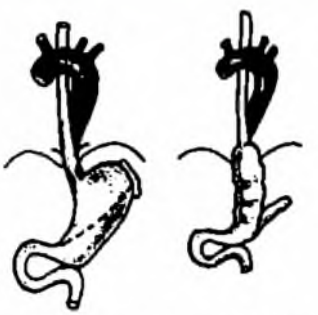
III- Carcinoma of the lower 1/3:

Partial oesophagogastrectomy :

Aim: to remove the upper 3/5 of the stomach usually with the spleen, tail of the pancreas and omentum together with the oesophagus up to 5 cm above the tumour and gastric pull up to the chest to be anastomosed with oesophagus .

Incision: 1 incision.

Lt. thoracoabdominal incision through the Lt. 8th rib bed.



Partial oesophagogastrectomy

Prognosis is very bad due to-

- Early spread
- High mortality of the operation (about 20 %)

Dysphagia

Definition:

Difficulty or just discomfort in swallowing.

Aetiology:

I- Oral causes:

1- Congenital: Cleft palate &ankyloglossia

2- Traumatic: wounds, corrosive, fracture mandible

3- Inflammatory: glossitis, stomatitis and ulcers.

4- Neuromuscular: trismus, tongue and cheek paralysis.

5- Neoplastic: tongue and maxillary carcinoma.

II- Pharngeal causes:

1- Congenital: Pharyngeal stenosis

2- Traumatic: wounds & corrosive.

3- Inflammatory: tonsilitis, pharyngitis and para-pharyngeal abscess.

4- Neuromuscular: cricopharyngeal achalasia, plummer Vinson syndrome & bulbar palsy.

5- Neoplastic: post-cricoid carcinoma.

III- Oesophageal causes:

A)In the lumen:

Foreign body.

B) In the wall.

1) *Congenital*: atresia, stenosis with or without tracheo-oesophageal fistula.

2) *Traumatic*: corrosives, instrumentation, and F.B trauma.

3) *Inflammatory*: non-specific (peptic ulcer) or specific (TB).

4) *Neuromuscular:* cardiac achalasia and myasthenia gravis.

5) Collagen diseases: scleroderma

6) *Neoplastic*: Leiomyoma, and cancer oesophagus.

C) Outside the wall (Compression dysphagia):

1) *Cervical part*: malignant thyroid & pharyngeal pouch.

2) *Thoracic part*: retrosternal goiter, enlarged LNs, dilated Lt. atrium, aortic aneurysm ,hiatus hernia & mediastinal tumours

3) *Abdominal part*: enlarged Lt. lobe of the liver.

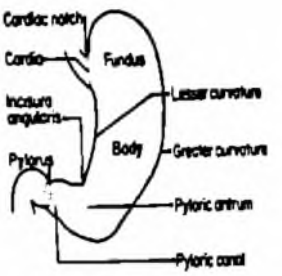
Stomach & Duodenum

Anatomy of the stomach

It is the widest part in the gastrointestinal tract, and acts as a temporary food reservoir beside its digestive and absorptive function.

Anatomical parts:

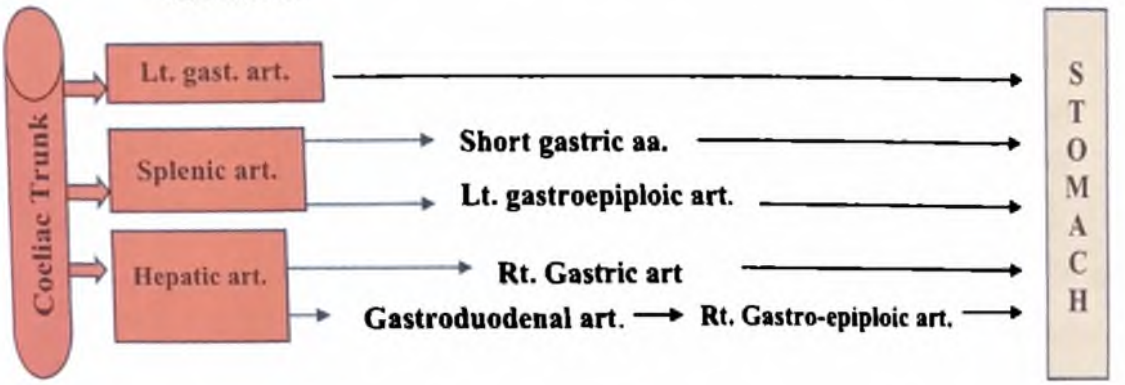
1) Cardiac end: connects stomach to esophagus at the level of T_{11}.
2) Fundus: the part lying above a horizontal line at level of cardia.
3) Corpus (body): the part lying between the previous horizontal line and a vertical line drawn at the level of incisura angularis.
4) Pylorus: the part lies distal to the previous vertical line.
   It is divided into pyloric antrum and pyloric canal, which controls passage of food to the duodenum.



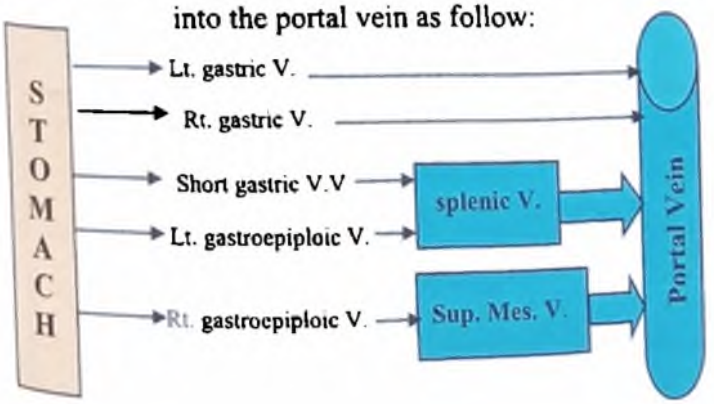
Anatomical parts

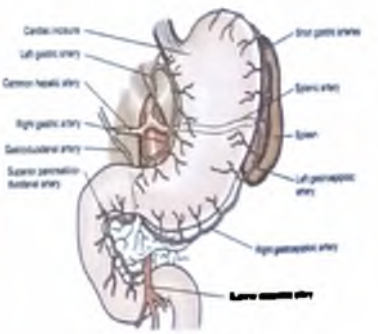
Blood supply:

A- Arterial: stomach derives its blood supply from the coeliac trunk (artery of the foregut) as follows.



B- Venous drainage: The veins draining the stomach are named after their arteries and drain into the portal vein as follow:



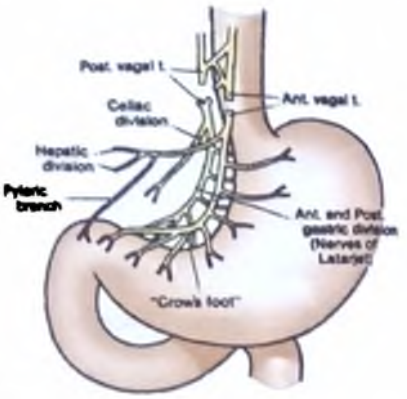


Arterial supply

Nerve supply:

A- Sympathetic: from the coeliac plexus around its arteries.

B-Parasympathetic: by both vagi which are named ant. & post. gastric nerves or ant. & post. nerves of Laterjet.



Nerve supply

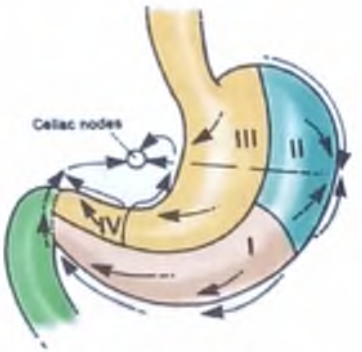
Lymphatic drainage:

The stomach is divided into 4 zones by 3 lines:

1) *Curved line* midway between the two curvatures.
2) *Vertical line* dropped from the Rt. edge of esophagus.
3) *Vertical line* dropped from the point junction of the upper 2/3 and lower 1/3 of lesser curvature.

Zones & Drainage:

1) Inferior gastric area (area I): drains into the inferior gastric (gastroepiploic) LNs. that drain into the subpyloric and retropyloric LNs., then to coeliac LNs.
2) Gastrosplenic area (area II): drains into the pancreaticosplenic LNs that drain into the celiac LNs.
3) *Superior Gastric area* (area III): drains into the paracardial LNs and coronary LNs, which drain directly to the coeliac LNs.



Lymphatic drainage

4) The pyloric area (area IV): drains into the suprapyloric LNs. that drains into the coeliac LNs

So the coeliac LNs are the central station for lymphatic spread of all cancers of the stomach. Further spread may occur from the coeliac LNs. in two main directions:

a) Along the lymphatics around the hepatic artery to LNs in porta hepatis. Also, along the ligamentum teres to the umbilicus → metastatic nodules (*Sister Joseph nodules*).

b) Along the lymphatics accompanying the thoracic duct to the left supraclavicular L.N.s (Virchow's LN) = *Troisier sign.*

The gastric bed

The structures related the posterior wall of the stomach constitute the gastric bed that forms the posterior boundary of the lesser sac. These structures are:

1- transverse mesocolon.
2- the body of the pancreas
3- the splenic artery.
4- the gastric impression of the spleen
5- the left kidney
6- the left adrenal
7- the diaphragm
8- the left gastric artery

Anatomy of the duodenum

It is the widest and most fixed part of the small intestine. It is 25 cm long with C shaped curve.

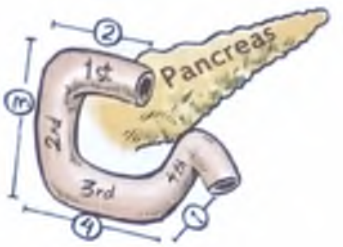
Anatomical parts:

1-*First part* is 2 inches long on the transpyloric plane (L1).

2-*Second part* is 3 inches long vertically downward.

3-*Third part* is 4 inches long on the subcostal plane (L3)

4-*Fourth part* is 1 inch long vertically upward.



Anatomical parts

Internal anatomy of the duodenum.

The second part has two papillae:

1- *Major duodenal papilla*: in its summit. opens the hepatopancreatic ampulla (ampulla of Vater) formed by fusion of the common bile duct (CBD) and main pancreatic duct.

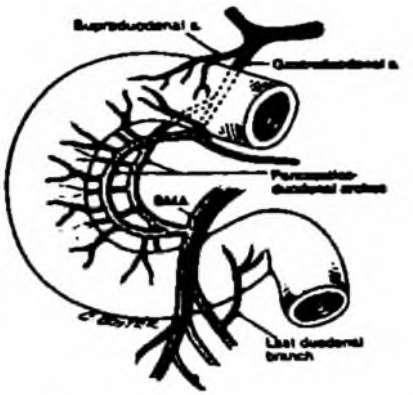
2- *Minor duodenal papilla*: in its summit, opens the accessory pancreatic duct.

Blood supply: (see the figure)

1) Arterial:

-The first part is supplied by branches from the supraduodenal, Rt. gastroepiploic & gastro-duodenal arteries.

-The other parts are supplied by branches from sup. & Inf. pancreaticoduodenal arteries.



Blood supply of the duodenum

2) Venous:

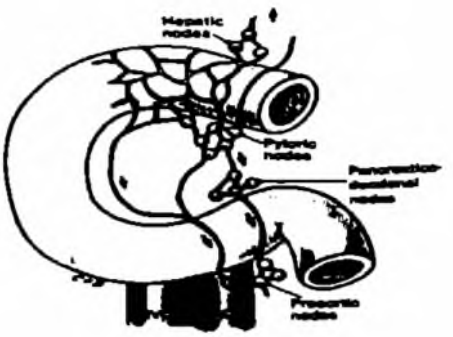
- The superior pancreaticoduodenal vein ends directly in the portal vein while the inferior pancreaticoduodenal vein drains to the sup mesenteric vein to portal vein.

Nerve supply:

By branches of the coeliac and sup. mesenteric nerves plexus reaching it around its arteries.

Lymphatic drainage:

Lymphatics pass first to a series of pyloric & pancreatico duodenal LNs lying between the duodenum and the head of pancreas. These LNs drain into the hepatic and sup. mesenteric LNs.



Lymphatic drainage

Applied Anatomy

1- *Prepyloric vein of Mayo* is an anastomotic vein between the right gastric and right gastro epiploic veins. It is an anatomical landmark of the pyloro-duodenal junction.

2- *Anterior and posterior nerves of Laterjet* end at the pylorus by what is called *Crow's foot* which is the parasympathetic supply to the pylorus

3- *Neve of Grassi* is a branch of the posterior vagus that originates at the level of the oesophago-gastric junction and supplies the gastric fundus. It is present in a good number of populations and may cause recurrent ulceration after vagotomy, so it is commonly termed by surgeons the criminal nerve of Grassi.

4- The stomach has a rich blood supply and has a dense sub-mucosal vascular plexus which is deficient on the lesser curvature that for this reason, it is the least vascular part of the stomach. Also, the first inch of the duodenum is supplied by the supraduodenal artery which is thought to be an end artery, so it is considered to be the least vascular part of the duodenum

5- The duodenum and the head of pancreas are considered as one surgical unit because they have common blood supply from the pancreatico-duodenal arches, so the duodenum can't be resected totally without resection of the head of pancreas and vice versa.

Histology

The gastric mucosa is lined with columnar epithelium and shows pits of gastric glands. The types of cells in the gland differ in different regions of the stomach as follow:

1- The cardiac gland area contains mainly mucous secreting cells.

2- The oxyntic gland area: The glands contains
   * Parietal (oxyntic) cells that secrete gastric acid and intrinsic factor.
   * Chief cells that secrete pepsin.
   * Mucous secreting cells.

3- The pyloric gland area: The glands contains:
   * G cells that secrete gastrin hormone that stimulates the parietal cells to secrete acid.
   * Mucous secreting cells.

Physiology

The stomach has the following functions

1) It is the first reservoir of food

2) It mixes the food with its acidic juice to form acid chyme . The acidity not only helps digestion of food but also sterilizes it

3) It begins digestion of carbohydrates & protein

4) It absorbs some nutrients and mineral

5) It regulates passage of the chyme to the duodenum in boluses

6) Parietal cells secret the intrinsic factor that is essential for absorption of vit. B_{12} in the ileum

Gastric motility

The stomach seems to have a pacemaker at its fundus near the greater curvature that initiates electric impulse at a rate of 3/min. peristaltic waves moves from the fundus through the body to the antrum where most of the food are propelled backward as the pylorus acts as a sphincter that opens only when the acid chyme is good mixed & emulsified . As other hollow organs emptying of the stomach is dependent on parasympathetic (vagal) supply that causes contraction of the wall and relaxation of the sphincter (pylorus)

Gastric secretion

The gastric secretion has the following phases

[1] The nervous phase: this phase is mediated by the vagus. Thinking, smelling or sight of food stimulate gastric juice secretion

[2] Gastric (humoral) phase: mechanical and chemical stimulation of the antrum release gastrin hormone that stimulate gastric secretion

[3] Intestinal phase: Presence of food in the intestine stimulates secretion of number of intestinal hormones that regulate gastric secretion.

The gastric juice contains

1) Mucous secreted by mucous secreting (goblet) cells

2) Hypochloric acid (HCL) secreted by parietal cells

3) Pipsinogen secreted by chief cells

4) Intrinsic factory secreted by parietal cells

5) Blood groups antigens: 75% of population (secretors) tend to secret their ABO blood group antigens into their gastric juice. Those secretor individual are less likely to develop duodenal ulcers than non-secretor

6) Electrolytes as Na+ , Cl - , H+, K+

The parietal cells have mainly three receptors for stimulation of the proton (H^+) pump:

1- Histamine H_2 receptors which are triggered by histamine.

2- Acetyl choline receptors which are stimulated through the vagus.

3- Gastrin receptors which are triggered by the gastrin hormone.

I- Congenital anomalies

1) - Congenital hypertrophic pyloric stenosis

Definition:

Congenital disease characterized by thick non relaxing pyloric sphincter (functional obstruction).

Pathology

Aetiology:

Immaturity of the ganglion cells in the pylorus → pyloric achalasia.

Pathology:

-*At the site:* marked hypertrophy of the circular muscle layer of the pyloric canal forming firm olive shaped swelling *(olive tumour)* which often projects into the duodenum like the uterine cervix in the vagina forming duodenal fornix around it.

-*Proximally*: the stomach at first hypertrophies to overcome obstruction then fails & dilates.

-*Distally*: the duodenum is normal and collapsed.

Complications:

1- Malnutrition & marasmus.

2- Dehydration and electrolytes deficit.

3- Hypochloraemic alkalosis.

Palpation of olive tumour

Incidence:

-Common in the *first born*, *mature male* baby usually with +ve family history, and breast fed.

Clinical picture:

Manifestations usually begin 2-3 weeks after birth:

-Vomiting is the cardinal symptom which is:

* Painless. *Projectile. * Postprandial (soon after meals).
* Bile free. *Post-vomiting hunger.

-Constipation.

-Visible peristalsis in the epigastrium from Lt. to Rt. especially soon after meals.

- Mass *(Olive tumour)* may be felt as firm ovoid mobile mass in the Rt. hypochondrium.

-Some cases come late with complications e.g. marasmus, dehydration & electrolytes deficit.

Investigations:

1-Ultrasound: increased thickness of the pyloric sphincter (> 3mm).

2- Gastrographin meal: It shows dilated stomach, narrow long pyloric canal (string sign), with shouldering of pylorus & delayed emptying.

Differential diagnosis:

It should be differentiated from other causes of persistent vomiting in neonates as:

1-Pylorospasm: no palpable mass and rapid response to conservative treatment.

2-Gastroenteritis: diarrhea excludes pyloric stenois, and the baby is toxic. Anorexia rather than hunger is usually present after vomiting.

3-Congenital duodenal atresia: vomiting starts immediately after birth and usually bile stained. X ray reveals *double bubble sign.*

4- Volvuolus neonatorum: vomiting starts few days after birth and is bile stained.

5-Intussusception: usually late in infancy. Attacks of colicky pain, and if a mass is felt it is sausage shaped, with *red currant jelly stool* on PR.

6- Increased ICT. (Intracranial hemorrhage): the baby is unhealthy, with CNS manifestations. Vomiting is not related to meals and usually bile stained.

Treatment:

I- Conservative ttt:

Indications

Mild cases with delayed onset e.g. at the age of 2 months.

It consists of:

1) Correction of fluid, electrolytes and acid base disturbance.

2) Eumydrine (atropine methyl nitrate) 1/10,000 conc. 2.5 cm ½ hour before each feed.

3) Frequent small feeds.

II - Surgery: ttt of choice.

* Pre operative preparation: -

- Nasogastric suction and gastric lavage with normal saline.
- Correction of hypochloraemic alkalosis by saline.

*Operation: *Ramstedt's operation* = pyloromyotomy

The seromuscular coat is divided longitudinally till the mucosa bulges. Incision should be stopped short of the *prepyloric vein of Mayo, so as* not to open duodenal fornix...

Ramstedt's operation

2- Duodenal atresia

Aetiology

Failure of canalization process.

Pathology

Site: Usually just below the major duodenal papilla.

Complications:

Dehydration, hypokalemia, & hyponatraemia.

Duodenal atresia

Clinical picture

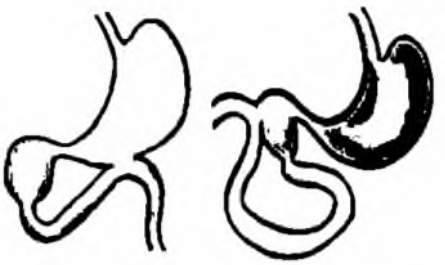
- Symptoms are dated since birth.
- Vomiting is the main symptom, persistent and *bile stained.*
- Constipation: failure to pass meconium.
- Visible peristalsis as pyloric stenosis but no mass is felt.
- Complications: severe dehydration and electrolyte deficit, with malnutrition.

Investigations:

- Plain X ray in erect position: shows the characteristic *(double bubble sign).*

Treatment:

- Pre operative preparation: fluids and electrolytes.
- Operation : bypass of the obstruction by either :
  1-Gastrojejunostomy not preferred as the blind duodenal pouch will cause repeated biliary vomiting.
  2- Duodenojejunostomy (side to side).
  3- Duodenoduodenostomy is the procedure of choice



Gastrojejunostomy Duodenojejunostomy

II- Trauma

1- Foreign bodies

Types:-

1) Ingested F.B. e.g. coins, pin etc.

2) Locally formed F.B. *(bezoars).*

A- Trichobezoars: from hair which may be eaten by children or mentally retarded girl.

B -Phytobezoars: from some plant fibers (eg. orange).

C- Shellac bezoars: in person swallowing furniture polish.

Treatment:

I) Conservative ttt: in most cases waiting for spontaneous passage in stool.

- Giving cotton wool in jam or honey sandwiches to wrap the F.B. (obsolete).
- Daily X ray abdomen and close observation in the hospital (stool examination).

II) Endoscopic removal: indicated in small ingested FB. in the stomach.

III) Operative removal:

Indications: 1- Large FB (bezoars). 2- Sharp or irregular FB.
3- Failure of conservative ttt. 4- Complications.

Procedure: Removal of the F.B through a gastrotomy.

2-Mallory Weiss syndrome

Definition:

Upper GIT bleeding due to longitudinal mucosal laceration at oesophagogastric junction.

Aetiology:

Violent retching & vomiting (90% of case) (increased gastric pressure against closed cardia).

Clinical picture:

-Common in alcoholic males over 50 y.

-The condition usually occurs after repeated attacks of retching (ineffective vomiting). After vomiting, profuse haematemesis follows.

-Severe cases may pass into shock then death.

Endoscopic picture

Investigations:

- Oesophagogastroscopy is diagnostic.

Treatment:

- Conservative ttt: most cases respond to sedation, IV fluids, and bl. transfusion.
- Surgical ttt: in resistant cases, by suturing the linear tear through a gastrotomy.

3-Volvulus of the stomach

Definition

Axial rotation of the stomach.

Aetiology

Predisposing factors:

A- Eventration of the diaphragm. B.-.Asplenia.

C- Heavy tumours on the greater curvature. D- Rolling HH

E- marked distension of the transverse colon. F- Lax ligaments

A- Organoaxial
B- mesentricoaxial

Pathology

Types: There are two types according the axis around which the stomach rotates

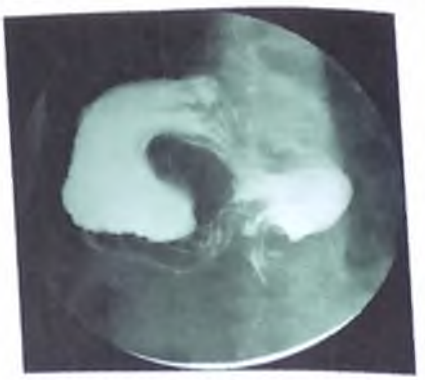
1-Organoaxial volvulus the stomach rotates around an axis from cardia to pylorus (common).

2-Mesentricoaxial volvulus the stomach rotates around axis from lesser to greater omentum.

Complications: in acute volulus

1- Shock.

2- Acute gastric necrosis.



Gastric volvulus

Clinical picture:

-The onset is sudden with (*Borchardt's triad):*

*Vomiting followed by retching & failure to vomit.

*Epigastric fullness.

*Failure to pass a nasogastric tube.

Investigations:

1-Plain X-ray in erect position: shows double bubble sign.

2-Barium meal: shows hourglass deformity of the stomach.

Treatment:

1-Nasogastric tube if it succeeds to pass to the stomach, it will reduce the volvuolus.

2-Surgical ttt:

-Urgent operation to reduce the volvuolus and correction of associated hiatus hernia.

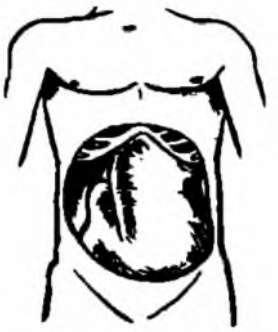
-Division of the gastrocolic ligament and fixation of stomach either to the duodenojejunal junction or to the anterior abdominal wall at three points.

Acute gastric dilatation

Aetiology:

A rare condition mostly due to sympathetic over-stimulation, which initiates a vicious circle.

Sympathetic irritation → Gastric atony → Gastric dilatation → Sympathetic irritation



Acute gastric dilatation at autopsy

The condition usually follows:

1) Abdominal surgery usually on another organ not the stomach.

2) Extensive injuries e.g. fracture spine or minor procedures e.g. catheterization.

Pathology:

-The stomach is hugely distended with fluid & gas (ingested, secreted & diffused from blood).

- The mucosa is congested with ulceration & petechial hage.

- The dilatation doesn't extend beyond the duodenum.

Complications and cause of death:

The same as paralytic ileus.

Clinical picture:

- The condition starts usually 2-3 days after operations.
- Effortless vomiting of large amount of brownish offensive fluid.
- Epigastric distension then huge abdominal distension.
- Rapid dehydration & shock in few hours.
- Tympanitic resonance and succusion splash may be detected.

Treatment:

I- Prophylactic: Wide nasogastric tube insertion before all abdominal operations.

II- Treatment of established cases:

1- Nasogastric suction through wide N/G tube.

2- Morphia.

3- IV. fluids and correction of electrolyte imbalance.

4- Prophylactic antibiotics.

Prognosis:

Untreated cases die rapidly.

III- Inflammation

Peptic ulcer

Definition: Ulceration of mucosa exposed to peptic digestion (acid and pepsin).

Acute peptic ulcers

Acute erosive gastroduodenitis

Definition:

Acute superficial ulceration (erosions) of the gastric and duodenal mucosa.

Aetiology:

I-Erosive ulcer: ulcerogenic drugs as aspirin, anti-inflammatory drugs & corticosteroids.

II- Stress ulcer: in stressful conditions e.g.

1- Prolonged hypotension in shock, & myocardial infarction due to mucosal ischemia.

2- Major burn (>35%) (Curling's *ulcer*).

3- Cerebral hemorrhage, trauma or operation (*Cushing's ulcer).*

Pathology:

The stomach is usually studded with multiple small oval or rounded superficial ulcers.

Complications:

1- Hemorrhage: varies from mild attack of melaena to severe attack of haematemesis.

2- Perforation: rare (< 5%).

Clinical picture:

The condition varies from mild dyspepsia to severe attack of haematemesis or perforation.

Investigations:

-Gastroscopy is diagnostic.

Treatment:

I- Prophylactic ttt - avoid the predisposing causes.

- H_2 receptor antagonists in risky cases.

II- Treatment of established cases:

1- Conservative measures: (usually succeeds):

a) Stop the offending drug.

b) Bl. transfusion.

c) H_2 receptor blockers.

d) Gastric lavage with iced cold saline.

2- Surgery:

Indications:

1) Failed conservative measures.

2) Complications that need urgent surgery e.g. severe hage with shock or perforation.

Procedures: Either

a) Vagotomy & pyloroplasty with suturing of bleeding ulcers.

b) Subtotal or total gastrectomy in massive gastric bleeding.

Chronic peptic ulcer

Aetiology:

Peptic ulcer results from the corrosive action of the acid & pepsin on vulnerable mucosa either due to increased acid secretion (duodenal ulcer) or decreased mucosal resistance (gastric ulcer).

Causes:

1) Duodenal ulcer = *hyperacidity*.

1- *Large parietal cell mass.*

2- *Increased vagal tone*: as in Tall Thin, Tense individuals (3 Ts).

3- *Endocrinal disorders* e.g. Zollinger Ellison syndrome (gastrinoma) & hyperparathyroidism.

4-*Liver impairment*: →decreased metabolism of gastrin and histamine

5-*Irritant food* → increase gastric acidity.

6-Infection with Helicobacter pylori (H. pylori): may be the principal cause. It infects the pyloric mucosa and splits urea with production of ammonia with rise in the pH → continued secretion of gastrin → hyperacidity.

-**Zollinger Ellison syndrome:** it is a syndrome of:
- Non B islet cell tumour of the pancreas or duodenum (gastrinoma).
- Gastric hypersecretion with elevated serum gastrin.
- Peptic ulcers in the duodenum and even in the proximal jejunum.
- Multiple complicated and recurrent ulcers.

-**H. Pylori** is gram-ve motile curved bacilli that infect only gastric mucosa. Hyperacidity induces gastric type metaplasia of the duodenal mucosa only then it becomes infected with H. pylori from adjacent antral gastritis.

II- Gastric ulcer = *diminished mucosal resistance*:

1) Duodeno-gastric reflux: due to disturbed pyloric sphincter mechanism that would cause bile induced atrophic gastritis. Smoking induces duodenogastric reflux.

2) Local ischaemia: may be due to atherosclerosis, thrombosis or vasculitis.

3) Malnutrition: deficiency of proteins (e.g. Madaras ulcer) or Vit A & C.

4) Trauma: by irritant food or ulcerogenic drugs.

5) Infection with H .pylori: It causes chronic gastritis and damages the mucous barrier with its enzymes. Also, it secretes cytotoxins.

Predisposing factors

Factors	Duodenal ulcer	Gastric ulcer
Age	20-40 y	40-60 y
Sex	♂ > ♀	♂ = ♀
Genetic	+ve family history more in bl. group O	+ve family history
Constitution	more in Tall Thin Tense persons	more in listless careless persons
Acidity	Hyperacidity	Normo or hypo-acidity
Motility	Hypermotility	Hypomotility

Pathology:

*Number: -Usually single, but, multiple ulcers may occur in Z.E. syndrome.

-Kissing ulcers are two duodenal ulcers, anterior & posterior.

*Sites:

1) *Duodenum* (commonest): duodenal ulcer (DU) usually lies in the 1st inch of the first part of the duodenum (duodenal bulb) due to the jet action of acid chyme.

-*Post bulbar ulcers* may occur in Z.E. synd., and commonly on the anterosuperior aspect.

-*Saddle shaped ulcer* may occur in Z.E. synd.

2) *Stomach*: gastric ulcer (GU) occurs over the lesser curvature (exposed to trauma).

3) *Jejunum* (in Z.E syndrome & stomal ulcer).

4) *Oesophagus* (in reflux oesophagitis).

5) *Ileum*: near Meckel's diverticulum containing ectopic gastric mucosa.

Ulcer bearing area

Size: Peptic ulcer is usually < 1 inch in diameter. GU is larger than DU.

Shape: Rounded or oval but usually, it is irregular due to fibrosis.

Edge: Sloping.

Base: Fibrosed serosa.

Floor: Granulation tissue over the serosa (damaged mucosa & musculosa).

Margin: Inflamed during activity, and the mucosal rugae converge toward it.

How to detect and localize the ulcer at operation?

1) *By inspection*: dull white serosa at the base of the ulcer with loss of the normal luster. Puckering of wall may be noticed.

2) *By palpation*: localized induration in the wall, the tip of the finger may enter into the ulcer niche.

3) *Stippling sign*: subserosal petechia over the ulcer base when the suspected area is rubbed by a piece of gauze.

4) *Gastrotomy*: if all criteria are -ve.

Complications:

1-Bleeding 2-Perforation 3-Cicatericial obstruction 4-malignancy (sec later)

Incidence:

DU is much commoner than GU (20 :1 in Egypt).

DU is common in young adult tall thin tense males. GU is common in middle age of both sexes.

Clinical picture:

Feature	Duodenal ulcer	Gastric ulcer
1- Pain		
-Site	Epigastric midline or slightly to the Rt.	Epigastric midline or slightly to the Lt.
-Type	Deep burning pain or stabbing.	Deep burning.
-Time	2 hours after meal or hunger pain (1/2 h. before next meal). It may awaken the patient during sleep (characteristic).	Immediately after or within 2 hours after meal.
-Precipitated by	Hunger.	Meals especially irritant food.
-Relieved by	Food and antacid.	Vomiting.
2-Vomiting	Rare unless pyloric stenosis occurs.	Frequent and may be self induced
3-Appetite	Good.	Good but patient is afraid to eat.
4-Body weight	Normal or increased.	Weight loss.
5-Periodicity	Marked , the attacks last for 2-6 weeks then free interval of 2-6 month, classically in spring & autumn, induced by work , worry and weather	Not so marked.
6- H&M	Common and melaena is more common than haematemesis.	Less common and haematemesis is more common than melaena.
7- Signs	Pointing sign with localized tenderness	More diffuse pain and tenderness.

Investigations:

I- Stool analysis: for occult blood

It is used as screening test, but it is not valid in Egypt due to prevalence of intestinal parasites.

II - Barium meal:

1-In duodenal ulcer: -The presence of ulcer niche (fleck of barium).
-Persistent trifoliate deformity of duodenal cap.

2- In gastric ulcer: -the presence of an ulcer niche on the lesser curvature and a notch on the greater curvature (muscle spasm).

Duodenal ulcer

III - Gastroduodenoscopy: the most diagnostic.

1- Makes a firm diagnosis and accurately detects the site & size of ulcer.

2- To detect malignancy in GU and takes 4 quadrants biopsy.

3- Diagnosis of the cause of bleeding in cases presented with H & M.

4- Follow up of cases under medical ttt or after surgical ttt.

Gastric ulcer

IV- Investigations for the cause:

1- Estimation of serum gastrin: to detect ZE. syndrome. Normal value is 50-100 pg /ml.

2- Investigations for H pylori:

* Invasive method: Endoscopic biopsy then culture or rapid urease test
* Non-invasive method: serological testing of IgG against H. pylori in patient serum.

Differential diagnosis:

-From other causes of dyspepsia with upper abdominal pain or discomfort e.g. chronic cholecystitis, chronic pancreatitis and functional dyspepsia.

Wilkie's triad: common association of chronic peptic ulcer, chronic appendicitis, and chronic cholecystitis.

Treatment:

I-Treatment of chronic duodenal ulcer

"The emergence of new potent and effective drugs makes the conservative ttt to get a very high success rate and the role of surgery to shrink".

I-Conservative treatment = medical ttt:

A- Rest: both mental and physical rest is required. Minor tranquilizers may be used.

B- Diet: 1) Avoid 4S (Smoking Spices, Spirits & Stimulants (coffee- tea-cola).

2) Avoid ulcerogenic drugs e.g. steroids & non steroid anti inflammatory drugs.

3) Avoid milk & milk products. They increase acid secretion by their protein &Ca^{-}

C- Drugs:

I) Drugs that inhibit or neutralize gastric acid:

1- Antacids: to neutralize the gastric acidity e.g:

i-Sodium bicarbonate: not preferred because it produces large amount of CO_2 that increase acidity (rebound hyperacidity) & may precipitate perforation.

ii-Mg trisillicate: may cause diarrhea.

iii-Aluminium hydroxide: may cause constipation.

-Combination of the last two types is an ideal antacid.

2- H2 receptors blockers: e.g. Cimitidine, Ranitidine, Famotidine.

They block the stimulant action of circulating histamine on the H_2 receptors of the parietal cells. They are effective drugs.

4- Proton pump blockers: e.g. Omeprazol, lanzoperazole.

It inhibits the proton (H^+) pump of the parietal cells reducing the gastric acidity by about 99%. It is very effective drug in a dose of 40 mg once gaily for 8 weeks.

ii) Cytoprotective drugs:

1) Pepsin antagonists: e.g. sucralfate.

- It binds to and coats the ulcer area, and has an antipepsin activity.
- It is given 1/2 hour before meal & bed time.
- It should not be used with antacids as the acidity is essential for its action.

2) Prostaglandins: e.g. PGE

In addition to its mucosal cyto-protective action, it reduces the acidity

iii) Drugs against H. pylori:

-Metronidazole (or tinidazole), tetracycline (or amoxicillin) & colloid bismuth are effective.

-Eradication of H. pylori would prevent the recurrence.

N.B. Triple therapy: combination of omeperazole, tinidazole and amoxicillin or clarithromycin is associated with high rate of cure and a low rate of recurrence

Conservative ttt should be continued for 6 months to 1 year

II-Surgical treatment:

*Indications: 1- Intractable ulcer or failure of medical ttt for 6 months.

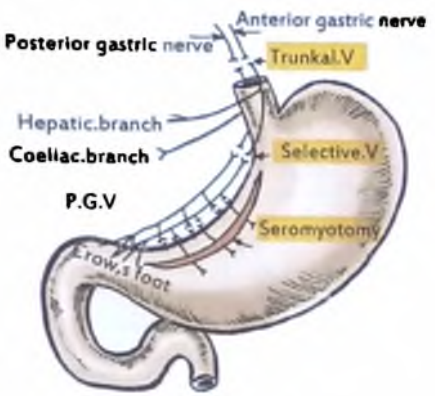
2- Complicated ulcer.

*Aim: to promote healing of the ulcer by reduction of the gastric acidity through one or more of the following procedures:

1- Vagotomy and drainage.

2- Subtotal gastrectomy.

3- Vagotomy and antrectomy.



Different types of Vagotomy

I) Vagotomy (vagal nerve section)

a) Trunkal vagotomy:

-It denervates the stomach & abdominal viscera.

-It causes pylorospasm & should be combined with drainage procedure to avoid gastric stasis that leads to foul eructation & recurrent ulceration

- Denervation of the intestines leads to chronic diarrhea, which may be disabling.
- Also denervation of the biliary tract leads to stasis and gall stones formation.

b) Selective vagotomy:

- It preserves the hepatic and coeliac branches of the vagi, thus, preserving the innervation of the other abdominal viscera to avoid the postvagotomy syndrome.
- Disadvantage: It still needs drainage procedure.

c) Super-selective vagotomy = Proximal gastric vagotomy (PGV) = Parietal cell vagotomy:

-It also preserves the antral branches of the vagi that are the nerve supply of pyloric sphincter (crow's foot). It does not need drainage procedure.

*Drainage procedures:

A-Pyloroplasty :(mikulicz)

6 cm longitudinal incision in the anterior wall of the pyloro-duodenal segment, which is closed transversely. It is more anatomical and physiological than gastrojejunostomy.

Pyloroplasty

B-Gastrojejunostomy:

Anastomosis (5-7 cm long) between the stomach & the proximal jejunum. It may be:

- Anterior (antecolic) or posterior (retrocolic).
- Isoperistaltic or antiperistaltic.

Indications: -when pyloroplasty is not feasible due to excessive scarring in the pylorus.

Anterior anterocolic Posterior retrocolic

Gastrojejunostomy

II) Subtotal gastrectomy

Principle: resection of up to 80% of the stomach.

Aim: 1- removing big part of parietal cell mass.

2- By removing the antrum, we remove the humoral phase of secretion.

Sub total gasterectomy

Anastomosis: by one of the following procedures:

1) Billroth II anastomosis: closure of the proximal pouch with side to side gastrojejunostomy (antecolic).

Billorth II anastomosis

2) Polya anastomosis: retrocolic end to side gastro-jejunostomy.

3) Hofmeister valve: partial closure of the stoma to form a valve that reduces dumping

Polya anastomosis

Hofmeister valve

NB. Billroth I operation (gastroduodenostomy) cannot be done in subtotal gastrectomy because:

a) The gap between the proximal and distal stump is wide.

b) The duodenum is the site of adhesions that interfere with *Kocherization* (mobilization) of the duodenum.

III) Vagotomy and antrectomy

Aim:

Removal of both nervous and humoral phase of acid secretion.

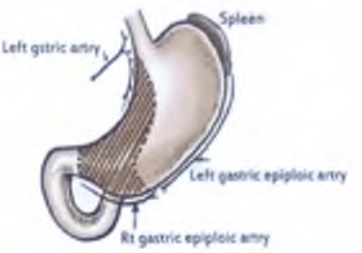
Procedure:

Vagotomy and removal of the distal 50% of the stomach.

Anastomosis:

Usually by Billroth I anastomosis, but may be by Billroth II.

-It has the same operative mortality (2%) as subtotal gastrectomy, but, it is more anatomical & physiological with little postoperative complications. In addition, it has a lower rate of recurrence (1%).



Antrectomy

Billroth I anastomosis

NB. If there is extensive fibrosis in the duodenum that would interfere with closure of the duodenal stump, do *Bancroft's technique*: that advises to do the distal cut in the healthy antrum not in the duodenum but with pealing of the antral mucosa in the distal stump down to duodenum to remove gastrin-secreting mucosa. Then close the resulting seromuscular tube and do Bilroth II anastomosis.

Choice of the proper procedure:

-If there is hyperactive vagus proved by Hollander's or chew and spit test, then do vagotomy and drainage e.g. pyloroplasty.

-If there is huge parietal cell mass proved by pentagastrin test, then do subtotal gastrectomy.

-If both, then do vagotomy and antrectomy.

-If there is weight loss → avoid gastrectomy i.e. super selective vagotomy.

II-Treatment of chronic gastric ulcer

1-Conservative ttt:

On the same lines as duodenal ulcer but with the following differences:

1-Specific drug (carbenoxolone sodium): a liquorice derivative that promotes healing of gastric ulcer through inhibition of prostaglandin dehydrogenase thus increasing prostaglandin level which is cytoprotective.

2-Conservative ttt is usually ineffective and recurrence is common so it should be continued only for 6 weeks, then gastroscope is repeated to assess healing. If no response or recurrence occurs, surgery is indicated.

II-Surgical ttt: is preferred:

Partial gastrectomy

Indications:

1- Failure or recurrence after medical ttt for 6 weeks.

2- Suspicion of malignancy.

Aim:

to remove the ulcer and ulcer bearing area + biopsy.

Procedures:

(1) Partial gastrectomy (hemigastrectomy):

Principle:

To remove about 65% of the stomach (distal 2/3).

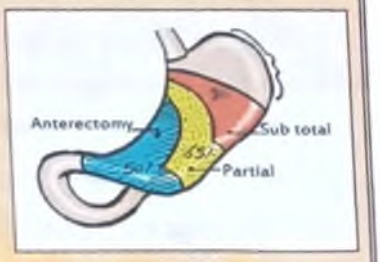
Anastomosis:

By Billroth I gastroduodenostomy, as the gap is small & the healthy duodenum can be mobilized (Kocherized) to bridge the gap.

(2) Subtotal gastrectomy

It may be used in high situated ulcer or combined GU & DU.

***In all types of gastrectomies:**



- The distal cut is the same i.e. in the 1st part of duodenum.
- The proximal cut starts at the same point 1 cm below the cardia on the lesser curvature to a variable point on the greater curvature according to the type of the gastrectomy:

* Subtotal 80% * Partial 65% * Antrectomy 50%

-The antrum with the whole lesser curve are removed in all types of gastrectomy.

Billroth I anastomosis has a great advantage over Billroth II anastomosis:

1. It is more anatomical (the gastric contents pass first to the duodenum),
2. It is more physiological as the following physiological processes occur normally in the duodenum
   a) Neutralization of gastric acid.
   b) Mixing food with bile and pancreatic secretion.
   c) Release of enterogastrone and cholecystokinin-pancreaticozymine (CCK-PZ) hormones.
   d) Absorption of iron and copper.
3. No afferent loop syndrome and minimal dumping (see below).

Complications of gastric operations

I- Intra-operative complications:-

1) Shock.

2) Haemorrhage (1ry).

3) Injury of important structures e.g. spleen, CBD, pancreas.

4) Difficult closure of the duodenal stump.

II- Postoperative complications

A- Early Complications:-

1- Reactionary hge: - commonly from the anastomotic line into the gut, and is detected by Ryle's tube and rarely via the drains.

2- Stomal obstruction: due to:

a- Postoperative oedema: common after Billroth I. It takes about 3-5 days to resolve.

b- Retrograde jejunogastric intussusception.

3- Leakage from anastomosis: it may cause fistula or subphrenic abscess.

4- Duodenal fistula (blow out): occurs around the 4th postoperative day. It results from rupture of the duodenal stump.

5- General post operative complications (after major abd. surgery) as:

a- Pulmonary complication: atelctasis, pneumonia, lung abscess.

b- Paralytic ileus.

c- Burst abdomen.

d- Infection: wound sepsis, subphrenic abscess.

B-Late complications:

***1-Postgastrectomy syndromes* 'include:**

a) Malnutrition syndromes:

I) Weight loss: due to reduced intake and malabsorption.It is treated by frequent small meals.

ii) Steatorrhea: lack of mixing of food with bile and pancreatic secretion.

iii) Diarrhea: due to steatorrhea or associated vagotomy.

iv) Iron & calcium deficiency: due to hypoacidity and duodenal bypass.

v) Megaloblastic anaemia due to malabsorption & lack of intrinsic factor.

b) Dumping syndromes:

Definition: a syndrome occurring after meals in patients who underwent surgical operations that destroy ,resect or bypass the pyloric sphincter due to rapid gastric evacuation.

I) Early dumping:

Aetiology: rapid evacuation of the hypertonic chyme into the jejunum increases the osmotic pressure in the intestine drawing fluid from the intravascular space causing hypovolaemia

Clinical picture: soon after meals (30min) there is:

Epigastric fullness, nausea, pallor, palpitation and sweating.

Treatment: most cases improve with time, and respond to conservative measures in the form of small frequent semisolid meals and parasympatholytic drugs & octreotide.

ii) Late dumping: Postprandial hypoglycemia.

Aetiology: rapid evacuation of large amounts of glucose →transient hyperglycaemia which induces intense insulin production with resultant *rebound hypoglycaemia*.

Clinical picture: 60-90 min after meal.

The patient develops symptoms of hypoglycemia e.g. epigastric emptiness, sweating, tremors, and palpitation.

Treatment: avoid CHO rich diet + olive oil before meal.

c) Afferent loop syndrome:

Aetiology: mechanical obstruction (e.g. kink) of the afferent loop with accumulation of bile & pancreatic secretion in it until obstruction is relieved with sudden gush of secretions into stomach.

Clinical picture: 15-30 min after meal, there is epigastric pain and distension which is relieved spontaneously and suddenly with vomiting of bilious fluid free of food.

Afferent loop syndrome

Conversion to Roux-en Y

Treatment: is usually surgical by conversion to Roux-en Y anastomosis.

d) Biliary gastritis:

Aetiology: reflux of bile into the stomach due to disturbed sphincter mechanism.

Clinical picture: postprandial pain, nausea, and vomiting of bilious gastric contents.

Treatment: Cholestyramine gives symptomatic improvement.

In resistant cases, conversion to Roux- en Y anastomosis is done.

2-Recurrent ulcer = anastomotic ulcer = stomal ulcer:

Aetiology:

I-Inadequate surgical procedure e.g. incomplete vagotomy, inadequate gastric resection. retained antral mucosa or inadequate drainage after vagotomy.

II-Persistent cause: Zollinger Ellison syndrome or hyperparathyroidism.

Pathology:

The same as the usual peptic ulcer except in the sites:

1- True anastomotic (stomal) ulcer: common on the efferent jejunal loop at the margin of the stoma, rare in the afferent loop as it is bathed with bile that neutralizes the acidity.

2- Gastric ulcer in the remnant pouch (rare).

Complications:

1-Haemorrhage: is common especially melaena, and massive hage may occur.

2-Perforation: acute perforation is rare, but chronic perforation (penetration) may occur into the colon to form gastrojejunocolic fistula.

Clinical picture:

Recurrence of ulcer symptoms within 2 years after operation, but, with some difference:

- *Pain*: -becomes more severe boring in nature and moves downwards and to the left.
- Vomiting: more severe and may be self induced.
- Haematemesis and melaena are more common.

Investigations:

1-*Barium meal:* shows ulcer niche (speck of barium) only in 50% cases.

2-*Gastroduodenoscopy*: most valuable

3-*Investigations to detect cause* e.g. Hollander's test to evaluate vagotomy and investigations for Z.E. syndrome & hyperparathyroidism.

Treatment:

1-Medical ttt: usually fails to achieve permanent cure

2-Surgery: according to the previous procedure.

a) *After vagotomy and gastrojejunostomy:*

-If incomplete vagotomy → complete vagotomy.

-If complete vagotomy → add antrectomy and Billroth I anastomosis.

b) *After partial or subtotal gastrectomy:*

-If recurrent duodenal ulcer (anastomotic ulcer) → add vagotomy.

-If recurrent: gastric ulcer → (higher gastrectomy).

d) *Zollinger Ellison syndrome:*

- If it is not detectable → total gastrectomy. (remove the target organ).

-If the tumor is detectable and resectable → excision.

3) Gastrojejunocolic fistula:

Aetiology:

penetration of anastomotic ulcer into the colon.

Clinical picture:

-The pain of the ulcer diminishes.

- Foul eructation and rarely true faecal vomiting.

-Watery diarrhea which may contain undigested food.

Investigations:

-Barium meal or enema to detect the fistula.

-Endoscopy is not valuable.

Gastrojejunocolic fistula

Treatment:

-After good colonic preparation, we resect the mass containing the fistula together with vagotomy and antrectomy or subtotal gastrectomy.

Resection anastomsis

4-Intestinal obstruction:

Aetiology

1- Retrograde jejunogastric intussusception.

2- Adhesive intestinal obstruction.

3- Bolus obstruction: where a bolus of unmasticated food (commonly a piece of orange pith) passes – in the absence of the pylorus – to the small intestine 'and lodges about two feet from the ileocaecal valve simulating gall stone ileus.

4- Herniation of intestine through the window in the mesocolon (Stammer's hernia)

5-Gallstones.

Due to stasis secondary to

1- Vagotomy → loss of the nervous phase of gall bladder motility

2- Duodenal bypass → lack of CCK PZ. → loss of the humoral phase of gall bladder motility

Complications of peptic ulcer

I-Acute complications

1-Haemorrhage.

2-Perforation.

II- Chronic complications:

1- Obstruction: pyloric stenosis and hour's glass or tea pot deformity.

2- Malignant transformation.

Bleeding peptic ulcer

Actiology:

A-Source of bleeding:

1-Mild bleeding (common), from the granulation tissue in the floor due to food trauma.

2-Moderate: due to erosion of a vessel in the base of the ulcer.

3-Severe fatal (rare) due to erosion of large extra-gastric vessel as the splenic or gastroduodenal art. or one of its branches.

Fatal haemorrahge

B) Predisposing factors:

Work, Worry, Weather and ulcerogenic drugs.

Incidence:

It complicates about 20% peptic ulcer cases.

Clinical picture:

-History of chronic ulcer dyspepsia in most cases.

-Mild cases present with chronic anaemia, which may be severe.

-Moderate cases may present with melaena alone with pallor.

-Severe cases with Pallor. Sweating and Faintness (hypovolaemic shock) then:

- Haematemesis (vomiting of coffee ground material or bright red blood)
- Then melaena (passage of black tarry offensive stool).

Investigations:

1-Gastroduodenoscopy: the most valuable:

2- Selective celiac angiography.

Differential Diagnosis:

From other causes of upper GIT bleeding especially bleeding oesophageal varices (see later).

Treatment:

1- Conservative treatment: usually successful. It includes:

1-Resuscitation: I.V. fluids, Bl. transfusion, and coagulants.

2-Nasogastric tube for

a) suction: to assess bleeding and prevent encephalopathy.

b) Lavage: with ice-cold saline to arrest bleeding and prepare for endoscopy. Iced citrated milk may be used.

3- I.V. H_2 receptor blockers and antibiotics.

4- Observation: vital signs, urine output, CVP and the amount of bleeding.

II-Interventional ttt:

- Endoscopic coagulation of the bleeding ulcer by Argon Laser or its injection with adrenaline or Histoacryl.

-Recently, selective infusion of bleeding vessels through angiography by Gelfoam embolization of the bleeding artery.

III-Surgical treatment:

Indications: 1-Failure of conservative ttt i.e. persistent or recurrent hage at hospital.

2- Old age (> 60 y) due to atherosclerosis and low cardiac reserve.

3- Long ulcer history, as dense fibrosis holds the bleeding vessels open.

Procedure: according to the general condition of the patient and source of bleeding.

1-Bad general condition: → stop bleeding & no more

Bleeding from the eroded bl. vessel at the base of ulcer is treated by under-running sutures.

2- Good general condition → definitive ulcer surgery.

a) Gastric ulcer → partial gastrectomy including the ulcer.

b) Duodenal ulcer → vagotomy and pyloroplasty with under-running suture the ulcer.

Under-running sutures

Prognosis: The prognostic factors:

1-Age of the patient: old age carries bad prognosis due to atherosclerosis & low cardiac reserve.

2-Amount of bleeding: the prognosis is worse in large amount of bleeding (> 6 units blood).

3-Time factor: moderate amount in a short time is worse than big amount in a long time.

4-Frequency: the 1st attack is usually not serious while the 3 rd attack is usually fatal.

Perforated peptic ulcer

Aetiology:

Endarteritis obliterans → ischaemia → sloughing of the base → perforation.

The same factors causing exacerbation of ulcer: worry, work, weather and drugs + Increase in the intra-gastric pressure e.g. heavy meal, straining & Na HCO_3.

Pathology: -

Site:

Commonly: on the anterosuperior aspect of the duodenum (DU) or anterior aspect of the lesser curve (GU). Rarely, a posterior ulcer may perforate into the lesser sac.

Common sites

Stages:

1-*Stage of perforation* = peritoneal irritation (2-6h).

Sudden escape of irritant gastroduodenal contents into the peritoneal cavity results in intense chemical irritation.

2-*Stage of peritoneal reaction* = lucid interval:

-The peritoneum reacts against this chemical irritation by secreting copious peritoneal fluid to dilute the gastroduodenal contents.

-This stage is invariably followed by diffuse septic peritonitis. The length of this stage depends upon many factors such as:

1-Size of the perforation: being longer with smaller perforation.

2-Fullness of the stomach: being shorter with full stomach.

3-General resistance of the patient: being longer in patient with good resistance.

4-Site of the perforation: longer in DU. than G.U. with better prognosis due to small size, high acidity, and empty stomach. Also, in duodenal ulcer, the contents pass along paracolic gutter to Rt. Iliac fossa, while in gastric ulcer, the contents pass freely to general peritoneal cavity.

3-Stage of diffuse bacterial (septic) peritonitis:

When body resistance fails, diffuse purulent inflammation occurs.

Leaking duodenal ulcer

Incidence:

-It complicates about 10 -20 % of peptic ulcer cases.

-It is common in middle age (40-60 y) male individuals.

Clinical picture:

-History of chronic ulcer dyspepsia is positive in about 75% of case.

1) Stage of perforation:

-Sudden severe agonizing pain in the epigastrium.

-Patient may pass into shock with pallor, sweating, subnormal temp & tachycardia.

-The board like rigidity is characteristic.

2) Stage of peritoneal reaction = lucid interval = (period of illusion) (6 h. later).

-The patient feels false improvement and looks better as pain diminishes, and temperature rises but:

* Tenderness and rigidity persist.
* Masked hepatic dullness by escaped gas may be detected.
* Shifting dullness may be elicited.

3) Stage of diffuse septic peritonitis 6 hours later, the pain returns severe with fever tachycardia and repeated vomiting.

-There is marked abdominal tenderness and rigidity with rebound tenderness.

-Abdominal distension with shifting dullness.

-Dead silent abdomen on auscultation.

Investigations:

1-Plain X ray of the abdomen or better chest in erect position→ gas under diaphragm. In negative cases, insufflation of 400 ml air through N/G tube may render them positive.

-If the patient can't stand, X ray may be taken in the Lt. lateral position → Gas in the axilla.

2-Barium meal: escape of barium through the perforation. (dangerous).

3- Abd. U/S: demonstrate turbid fluid in the peritoneal cavity.

Gas under diaphragm

Differential diagnosis:

From other causes of acute abdomen (see later) especially acute pancreatitis.

Treatment:

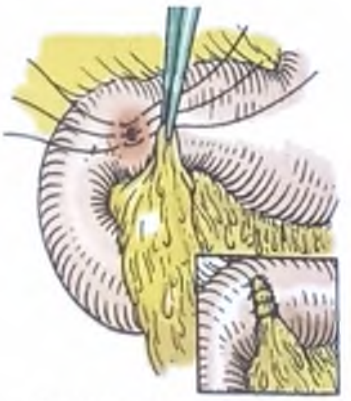
I-Resuscitation: IV fluids, bl. trans., antibiotics & N/G suction.

II- Conservative ttt = Non-operative management:

- Indicated only in poor risk patients unfit for surgery.
- It is not recommenced for routine use. It includes the resuscitation as before with close observation for the hope of spontaneous closure and localization of infection.

III– Surgical ttt: The ttt of choice: (open or laparoscopic)

It should be done as soon as patient is resuscitated.



Graham's Omental flap

Procedure: depends upon the general condition of the patient:

1-Bad general condition = simple closure and no more.

Simple closure with 3 interrupted full thickness sutures over *Graham's omental flap.*

2-Good general condition = definitive ulcer surgery either vagotomy with drainage in D.U., or partial gastrectomy in GU.

N.B. Gastric ulcer should be biopsied in all cases to rule out malignancy.

Prognosis:

-The mortality rate about 15%.

3-Chronic obstruction

1-Chronic Gastric outlet (pyloric) obstruction:

Aetiology:

Cicaterization of duodenal ulcer.

Pathology:

1-The pylorus: cicatrized and stenosed.

2-The stomach: at first hypertrophies then it fails and dilates. The hugely dilated stomach may reach the pelvis.

3- The small intestine: free and collapsed.

Complications:

1- Metabolic hypochloraemic alkalosis due to chronic H^+ & Cl loss.

2- Electrolytes deficit: hyponatraemia, & hypokalaemia.

3- Dehydration and prerenal anuria.

4- Loss of body weight and hypovitaminosis.

Clinical picture:

-History of ch. ulcer dyspepsia is almost always + ve.

-Epigastric distension especially after meal.

-Vomiting:

* Projectile. *not bile stained. * Foul odour.

*contains old food. *relief occurs after it.

-Progressive constipation.

-Visible peristalsis in the epigastrium from left to right

-Succusion splash may be elicited "two hours after meal".

-Picture of complications e.g. dehydration, tetany & electrolytes deficit.

Investigations:

1) Saline load test: 700 ml saline is infused through Ryle's tube. After ½ h, if > 350 ml is aspirated → obstruction is diagnosed. It is useful for follow up.

2-) Barium meal: it reveals:

i- Hugely dilated stomach (J. shaped) which may reach the pelvis.

ii- Fluid level in the stomach giving *soup plate appearance.*

iii- Delayed evacuation of barium. Remnants of barium may be still in the stomach after 24 h.

Soup plate appearance

3) Endoscopy:

-Fails to pass to the duodenum.

-Excludes malignant obstruction.

4) Bl. chemistry: to detect electrolytes and acid /base imbalance.

Differential diagnosis:

From other causes of gastric outlet obstruction (see later) especially malignant obstruction.

Treatment:

I-Conservative treatment:

Indications:

1-For mild cases (pylorospasm).

2- Cases unfit for surgery.

It includes:

-Nasogastric suction through wide N/G tube with gastric lavage to:

a-decompress the stomach and regain its tone.

b-allow pyloric oedema and spasm to subside.

-Perform saline load test to provide a baseline for later comparison.

-Correction of electrolytes or fluid deficit through I.V. fluids.

-Total parenteral nutrition for chronic malnourished cases.

-72 hours later, saline load test is repeated. If it shows good improvement, the N/G tube is withdrawn and liquid diet begins followed by gradual resumpsion of solid food.

II- Surgical treatment: in most cases:

-After proper preoperative preparation.

-The procedure depends upon the age and the general condition of the patient:

a) Old patient with bad general condition → relieve obstruction and no more or gastrojejunostomy alone (Pyloroplasty cannot be done due to marked fibrosis).

b) Young patient with good general → definitive ulcer surgery → vagotomy and gastrojejunostomy or subtotal gastrectomy.

II-Hourglass and teapot stomach.

Aetiology:

Cicatrized gastric ulcer.

Clinical picture & complications:

As pyloric obstruction.

Hourglass stomach **Teapot stomach**

Investigations:

1- Barium meal shows the persistent deformity.

2- Endoscopy.

Treatment: is mainly surgical:

I-Bad general condition: -

Gastrojejunostomy anastomosing the proximal pouch to the jejunum.

Alternatively, gastro-gastrostomy anastomosing the two pouches.

Gastro-gastrostomy

II-Good general condition:

Partial gastrectomy removing the distal pouch and the isthmus.

Causes of gastric outlet obstruction

I-In the lumen: -

F.B impaction.

II-In the wall:

1- Congenital: hypertrophic pyloric stenosis.

2 Traumatic: -Corrosive injury.

-After simple closure of perforated DU.

3-Inflammatory: - Cicatrized duodenal or prepyloric ulcer (commonest).

-Specific: T.B, syphilis or Crohn's disease (rare).

4-Functional or neuromuscular: pylorospasm or achalasia of the pylorus.

5- Neoplastic: carcinoma (second common).

IV- Compression form outside:

Bands, cancer head of pancreas, lymph nodes.

4-Malignant transformation

It occurs only in gastric ulcer. Duodenal ulcer is immune against malignancy.

Incidence: 5-10% of gastric ulcers turn malignant

IV- Tumours

Benign tumours

I- Adenoma: commonly at the antrum. It may be sessile or pedunculated. It may turn malignant

II- Leiomyoma, fibroma & lipoma: They may cause chronic bl. loss or serious acute bleeding

Gastric carcinoma

Aetiology:

I- Predisposing factors:

1-Environment: common in urban areas.

2-Race: common in Japanese.

3-Diet: -carcinogenic hydrocarbon (e.g. 3, 4 benzpyrine) in smoked meat & fish & in tobacco

- Spirits.

4-Heredity: - more in individual with + ve family history and those of bl. Group A.

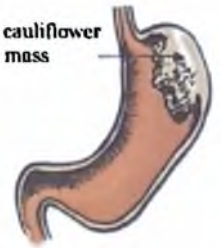
II- Precancerous conditions:

	Atrophic gastritis (H. pylori)		Pernicious anemia.
3As	Achlorohydria	**3Ps**	Peptic ulcer (ch. gast. ulcer).
	Adenomatous polyp		Post gastrectomy & post vagotomy.

Pathology:

Sites: Pylorus = 65%. Body = 25%.

Fundus = 5%. Diffuse = 5%.



Fungating mass

MAC: Lauren's classification

I- Intestinal gastric cancer (80%):

- It occurs on top of intestinal metaplasia of gastric mucosa.
- It occurs mostly in the fundus & the body of the stomach.
- It takes one of the following two forms:

1-Fungating (polypoid) mass (40%):

More in the body and fundus.

2-Ulcerative (malignant ulcer) (40%), the most malignant:

More in the pylorus – (mention its characters).

Linitis plastica

II- Diffuse gastric cancer = Infiltrative type (20%):

-Usually in the pylorus.

- It infilterates the wall of the stomach without forming a considerable mass.

-It may extend to involve all parts of the stomach with diffuse fibrosis converting it into narrow tube " *linitis plastica* " or "*leather bottle stomach* " .

MIC:

1-Adenocarcinoma: (95%):

a- Columnar cell adenocarcinoma: cells are arranged in acini.

b- Spheroid cell adenocarcinoma: rounded cells with loss of the acinar pattern.

c- Mucoid (colloid) carcinoma: increased secreting power of the cells → distension of the cells with mucous pushing the nucleus to one side giving *the signet ring appearance*. Rupture of some cells forms lakes of mucin among the acini.

Signet ring appearance

2-Squamous cell carcinoma: due to spread of oesophageal carcinoma to the cardia.

Spread:

1-Direct spread: A- Intrinsic: - 5 cm beyond the palpable edge.

B- Extrinsic: to liver, pancreas, transverse colon, spleen, diaphragm.

II- Lymphatic spread: (most important.) by both embolization and permeation. (See anatomy)

III- Blood spread: Usually to the liver, rare to the lung and bone.

IV- Transperitoneal spread: In tumours reaching the serosa, malignant cells detach and are implanted on omentum, peritoneum, & abdominal organs forming malignant nodules and ascites, It may give rise to *Krukenberg's tumors* in females and a rectal shelf in males.

Complications = causes of death:

1- Athenia and cachexia.

2- Perforation.

3- Obstruction: of the cardia or the pylorus.

4- Bleeding.

5- Distant metastasis.

Transperitoneal spread & Krukenberg's tumour

Incidence

-The commonest malignancy of the GIT.

-It is common in the Far East e.g. Japan.

- It is common in old age males.

Clinical picture: -

1-Dyspeptic group: (the commonest). It must be differentiated from dyspepsia of peptic ulcer by:

a) Short duration.

b) Pain is continuous with little relation to meal and not relieved by vomiting or drugs.

c) Vomiting is spontaneous.

d) Loss of appetite especially to meat.

2-Insidious (غادر) group:

Vague symptoms (3A): Anaemia, Anorexia and Asthenia

3-Obstructive group:

The patient may present with symptoms of oesophageal or pyloric obstruction.

4-Latent (كامن) or silent group:

The disease may remain silent till late or distant manifestations. e.g.:

a) Jaundice and liver metastasis or *Sister Joseph nodules.*

b) Enlarged Virchow's gland (*Troisier sign).*

c) Migrating thrombophlebitis of the superficial leg veins (*Trousseau's sign*).

d) Malignant ascites and *Krukenberg's tumour.*

5-Complications e.g. bleeding or perforation may be the first presentation.

Investigations:

I-Laboratory:

1-Occult blood in stool with anaemia (may be pernicious anaemia).

2-Gastric function tests: show hypo- or achlorohydria in most cases.

II- Radiological: =Barium meal or double contrast meal:

1-Fungating tumor: shows irregular filling defect usually in the body and fundus. Fundal tumours may need examination in Trendlenberg's position.

2-Ulcerative tumor: it shows large ulcer niche outside the ulcer bearing area and shows *Carman's meniscus sign* (crescentic barium shadow around the elevated edge of the ulcer). Also, mucosal rugae fade away from the ulcer.

3-Infiltrative tumour: it shows irregular narrowing of the pyloric canal with minimal proximal dilatation. In linitis plastica, it shows narrowed rigid tube.

III-Gastroscope: (most valuable):

Detection of the site, extent, and to take biopsy.

IV- Endoscopic U/S: It has the advantage of detecting the degree of infilteration of the wall and perigastric extension.

V- Estimation of carcinoembronic antigen (CEA) which is a tumour marker elevated in most of GIT tumors.

Carman's meniscus sign

VI- Exfoliative cytology:

VII- Investigations to detect metastasis: CT, MRI & abdominal U/S.

Differential Diagnosis:

From other causes of dyspepsia especially peptic ulcer (see the clinical picture)

Treatment:

1-Operable cases

A-Preoperative preparation: Improving the general condition of the patient may need total parentral hyperalimentation.

The rule: regardless of the site of the tumour:

-The distal cut should be beyond the prepyloric vein of Mayo.

-The proximal cut should be 3 inches above the palpable edge of the tumour.

B-Procedure: radical resection according to the site of growth:

1-Carcinoma of pylorus and antrum: -

Radical subtotal gastrectomy:

Aim: to remove:

1- About 85% of the stomach. 2- Lesser and greater omenta.

3- Spleen and tail of the pancreas. 4- First inch of the duodenum.

Anastomosis is done by Billroth II gastrojejunostomy.

Loop Oesophagojejunostomy

2-Carcinoma of the body and diffuse carcinoma:

Radical total gastrectomy.

Aim: Removal of the whole stomach together with the same structures removed in radical subtotal gastrectomy.

Anastomosis: is done either by:

-Oesophagojejunostomy: loop anastomosis with jejuno-jejunostomy or better Roux en Y anastomosis.

-Jejunal loop interposition (better).

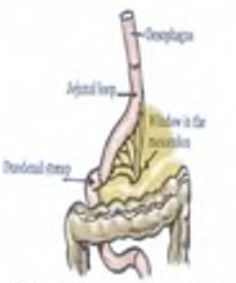
Roux en Y Oesophago-jejunostomy

3-Carcinoma of the fundus and cardia:

Oesophagogastrectomy (thoracoabdominal):

Aim: to remove the lower oesophagus in addition

Anastomosis: by Roux –en- Y oesophagojejunostomy.



Jejunal loop interposition

II- Inoperable cases:

Criteria of inoperability: See before:

I- Surgery

A-Resectable: palliative total or subtotal gastrectomy to avoid complications e.g. Hage, perforation – etc.

anterior gastrojejunostomy

B-Irresectable: -

Anterior gastrojejunostomy, or preferably, *Divine's exclusion gastrojejunostomy* is done.

II- Chemotherapy.

III- Radiotherapy.

Prognosis:

It is very bad due to:

1) Late presentation.

2) Early lymphatic spread.

Divine's exclusion gastrojejunostomy

Japanese concept of tiers of lymph nodes

They suggest that lymph nodes draining the stomach are arranged in tiers around it to which cancer may spread progressively

1- First tier (N_1): comprises LNs closest to the stomach.

2- Second tier (N_2) comprises LNs around the coeliac trunk and along its three branches.

3- Third tier (N3) comprises more distant LNs in the root of mesentry, retropancreatic, along the middle colic artery and para-aortic LNs.

*Japanese classify gastric resection according to radicality into:

R1: Resection includes lymph nodes mentioned in N_1

This is carried out by removal of the stomach with greater and lesser omenta

R_2: Resection includes lymph nodes mentioned in N_2

It necessitates removal of the spleen and body and tail of pancreas in addition to R_1

R3: Resection includes lymph nodes mentioned in N_3

It may necessitate partial colectomy, hepatic lobectomy, subtotal pancreatectomy or even pancreaticoduodenectomy

If R< N: palliative resection

If R= N: relatively curative resection

If R> N: curative resection.

Sarcomas of the stomach

I- Gastrointestinal Stromal cell tumour (GIST) previously known as leiomyosarcoma.

II- Lymphomas: Hodgkin or more commonly non-Hodgkin lymphoma previously known lympho-sarcoma. They are commonly associated with H pylori infection

UPPER GIT BLEEDING

Definitions

Bleeding originating above the ligament of Treitz usually presents as:

- **Haematemesis** = vomiting of bright red or coffee ground blood due to conversion of hemoglobin to methaemoglobin by the action of gastric acid (time factor is important).
- **Melaena:** (passage of black, offensive& tarry stool) due to action of intestinal enzymes on Hb (haematin). It can be produced by as little as 50ml of blood originating above the ileocaecal valve with intestinal transit time > 8 hours .
- **Occult blood in stool:**

  Bleeding < 50ml /day is not detected clinically, but detected by special tests as ***Benzidine test***
- **Haematochesia**: = passage of bright red per rectum.

  Massive upper GIT bleeding (e.g. > 1.5 liters) accelerates intestinal peristalsis, so blood passes unchanged (bright red) in stool.

Causes:

I-General causes:

Hypertension, bleeding tendency, & uremia.

II-Local causes:

A-Oesophageal causes:

1- Esophageal varices (the commonest cause in Egypt).

2- Mallory Weiss Syndrome.

3- Reflux oesophagitis and peptic ulceration.

4- Cancer esophagus.

B-Gastro duodenal causes:

1- Acute gastritis and multiple gastric erosions (2nd common).

2- Bleeding peptic ulcer (third commonest).

3- Peutz Jegher's syndrome: multiple GI hamartomas + mucocutaneous pigmentations.

4-Vascular malformation e: Dielafoy's disease (submucosal gastric vascular malformation)

4- Cancer stomach.

III-False haematemesis:

Vomiting of blood ingested after bleeding from mouth, nose or pharynx.

Diagnosis:

I-Clinical

A- From the history

1- History of Bilharziasis, previous sclerotherapy→ bleeding Varices.

2- History of major stress as major burn, stroke.etc → stress ulcers.

3- History of drug intake (ulcerogenic in gastric erosion).

4- History of dyspepsia in ch. peptic ulcer.

5-History of repeated vomiting or retching before haematemesis→ Mallory Weiss syndrome

6- History of hypertension, bleeding tendency, and renal troubles.

B- Examination:

1-General: -For vital signs (shock), anaemia.

-For general signs of the cause e.g.

*Jaundice, spider naevi, & fetor hepaticus in esophageal varices.

*Major insult in erosive gastritis.

*Uraemia manifestations.

2-Abdominal:

-Hepatosplenomegaly + ascites → bleeding varies.

-Epigastric tenderness →peptic ulcer and acute gastritis.

-Epigastric mass or evidence of metastasis →cancer.

II- Investigations:

A- Laboratory:

1- Hb %, hematocrite, bleeding time, & coagulation time.

2- Liver & kidney function tests.

3- Tests for occult bl. in stool.

B- Upper GIT endoscopy:

The most valuable investigation both diagnostic & therapeutic.

C- Coeliac Angiography:

In difficult cases with persistent bleeding.

Treatment:

1-Hospitalization. 2-IV. fluids. 3-Blood transfusion.

5-Morphia (not in LCF.). 6-H_2 receptor blockers.

7-Definitive treatment of the cause after diagnosis.

INTESTINES

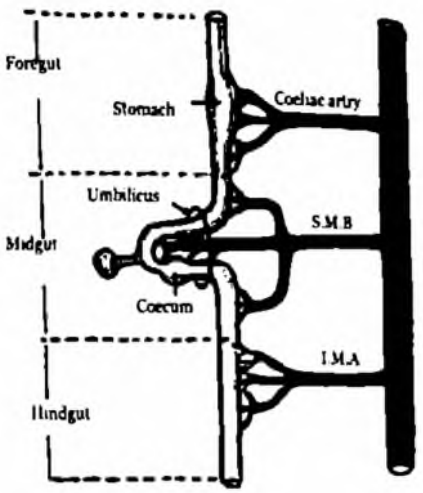
Embryology

The developing gut consists of three parts:

1- **Foregut**: gives rise to stomach and proximal duodenum .It is supplied by the coeliac trunk.

2- **Midgut:** gives rise to distal duodenum (distal to the major duodenal papilla), small intestine, proximal colon up to the junction between the proximal 2/3 and the distal 1/3 of the transverse colon .It is supplied by the sup. mesenteric artery.

3- **Hindgut**: gives rise to the distal colon, rectum and the upper part of the anal canal down to the dentate line. It is supplied by the inf. mesenteric artery.



The development of the gut

Stages of development:

1- Stage of physiological herniation (4th week) :

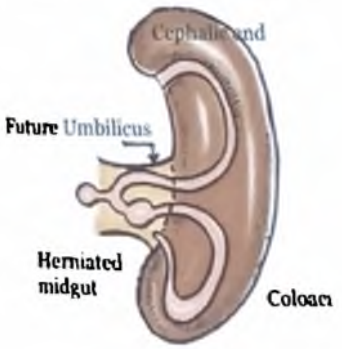
The growth of the gut is faster than the expansion of the coelomic cavity so, a part of the midgut loop is herniated to outside in the umbilical cord.

2- Stage of rotation and reduction of the herniated loop (5th - 11th week):

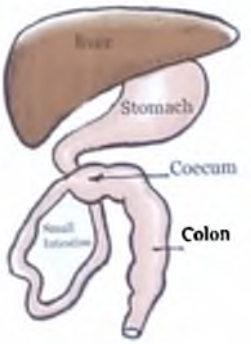
The coelomic cavity enlarges so, the herniated loops begin to enter again to the abdomen with anti-clockwise rotation.

3- Stage of intra-abdominal rotation and absorption of mesentry (after 11th week):

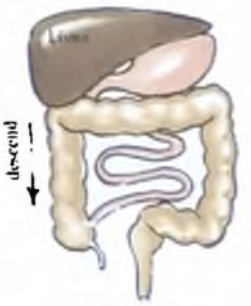
The midgut completes the 360° anti-clockwise rotation so, the caecum descends to the right iliac fossa with subsequent absorption of mesentry of the ascending and descending colon.



Stage I



Stage II



Stage III

Anatomy of the small intestines

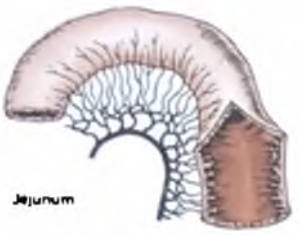
It is about 6 meters long in adult. It extends from the 4th part of duodenum to the caecum. It is the only part of GIT, which cannot be completely resected.

Begins: at the duodenojejunal junction which is fixed by ligament of Treitz attaching it to the Rt. crus of diaphragm.

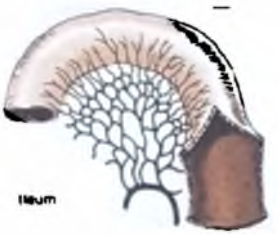
Ends: at the ileocaecal junction.

-It has a long mesentery that attaches it to the post. abdominal wall so, it is freely mobile.

-The root of mesentry extends obliquely across the post. abd. wall from the duodenojejunal junction downward to the right to the ileocaecal junction (15 cm long).



A loop of jejunum



A loop of ileum

Parts:

The upper 2/5 is the jejunum and the lower 3/5 is the ileum:

Item	Jejunum	Ileum
- Thickness – lumen	Thick wall & wide lumen.	Thin wall and narrow lumen.
- Arterial arcades	- One or two rows only.	- More complex (numerous).
	- apparent windows.	- Windows hidden by fat.
- Mucosal folds	- Numerous.	- Fewer.

Blood supply:

Arterial: by jejunal and ileal branches of the superior mesenteric artery.

Venous: veins drain into sup. mesenteric v. which unites with splenic v. to form portal v.

Nerve supply:

-*Sympathetic:* from the lesser and greater splanchnic nerves.

-*Parasympathetic:* from the right vagus nerve.

Both reach the intestine around the vessels.

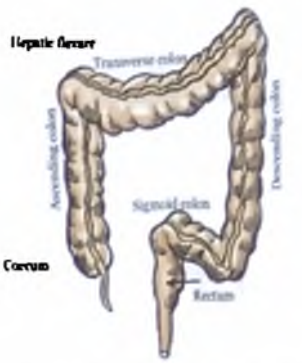
Lymphatic drainage:

Lymphatic channels from the intestines (intestinal lacteals) drain through three groups of LNs. in the mesentry, small sized LNs. on the jejunal and ileal branches and large sized LNs. around SMA. Lacteals fuse forming intestinal lymph trunk, which ends in the cisterna chyli.

Anatomy of the large intestines" the colon"

- Anatomically, the colon is divided into caecum, ascending colon, transverse colon, descending colon and sigmoid colon.
- It is about 180 cm long extending from the end of ileum to the rectum. The lumen gets narrower distally.
- The ascending & descending colons are fixed while the transverse & sigmoid ones have a mesentry so, they are mobile.
- Traditionally, it is divided into:

-**Rt. colon**: comprises caecum, ascending colon, hepatic flexure and proximal transverse colon.



Anatomical parts of the colon

-**Lt. colon:** comprises distal transverse colon, splenic flexure, descending and sigmoid colon.

- Surgically, the colon is composed of four surgical units according to its blood supply:

1- The Rt. colon supplied by the ileocolic and the Rt. colic arteries.

2- The transverse colon (middle two 2/4) supplied by the middle colic artery.

3- The Lt. colon supplied by the Lt. colic artery.

4- The sigmoid colon supplied by the sigmoid arteries.

The four surgical units

How to differentiate large from small intestines?

The colon is characterized by:

1- Taenia coli, which are the longitudinal muscle fibers, gathered into three longitudinal bands.

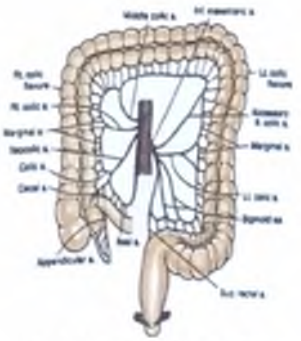
2- Haustrations As the length of the taenia is shorter than the length of the colon so, it becomes sacculated.

3- Appendices epiploicae: They are subserosal aggregations of fat.

Blood supply:

1- Sup. Mes. A
- Ileocolic a. → terminal ileum, caecum & appendix.
- Rt. colic a. → Ascend. colon, & hepatic flexure.
- Mid. colic a → proximal 2/3 of transverse colon.

2- Inf. mes. A
- Lt. Colic a
  - → Distal 1/3 of transverse colon.
  - → Splenic flexure & descending colon.
- Sigmoid aa. → Sigmoid colon

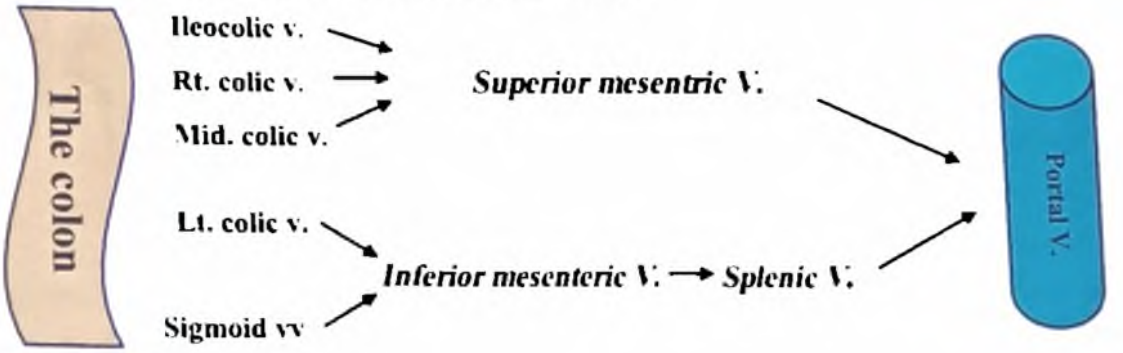


Blood supply of the colon

Marginal artery of Drummond: is formed from anastomotic branches of the ileocolic, Rt. colic, middle colic, Lt. colic and sigmoid arteries. It runs along and 2-3 cm away from the inner side of the colon. From the marginal artery, straight vessels (vasa recta) pass to supply the colon.

2-Venous drainage:

The veins draining the colon are named after their arteries:



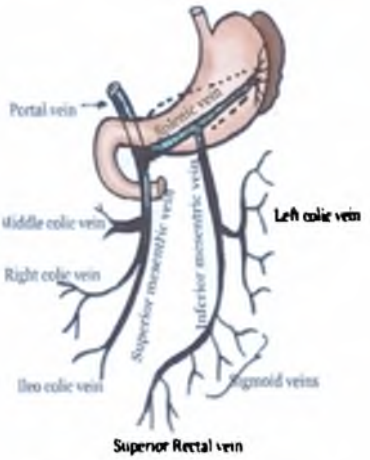
Nerve supply:

I-Rt. colon down to the junction bet. Proximal 2/3 and distal 1/3 of the transverse colon.

- *Sympathetic:* from sup. mesenteric ganglion (plexus).
- *Parasympathetic*: from the vagus.

II - Lt. colon (distal to the previous limit).

- *Sympathetic: from inf. mesentric ganglion (plexus).*
- *Parasympathetic: from pelvic splanchnic nerves (S2,3,4).*



Venous drainage of the colon

Lymphatic drainage:

The lymph nodes draining the colon are divided into 4 groups:

1- *Epicolic lymph nodes* on the wall of the colon.

2- *Paracolic lymph nodes* on the vascular arcades.

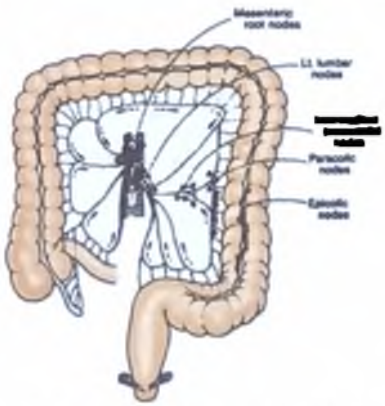
3- *Intermediate group of lymph nodes* on the main branches of the sup. and inf. mesentric arteries i.e. ileocolic, right colic, middle colic, left colic and sigmoid arteries.

4- *Main (central) group of lymph nodes* lies on the main trunks of the sup & inf. mesentric arteries.

Lymphatics pass between them with that sequence.

-Lymphatics of the Rt. colon end in the sup mesentric LNs.

Lymphatics of the Lt. colon end in inf. mesentric. LNs

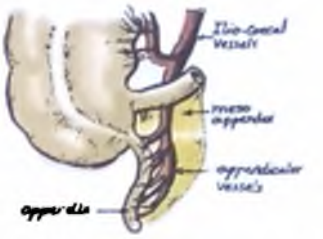


Lymphatic drainage of the colon

Anatomy of the vermiform appendix

It is a blind tubular diverticulum of the caecum about 7.5 cm long.

The three-taenia coli converge at the base of the appendix 1 inch below the ileocaecal junction where they form a continuous longitudinal muscle coat of the appendix.



Blood supply of the appendix

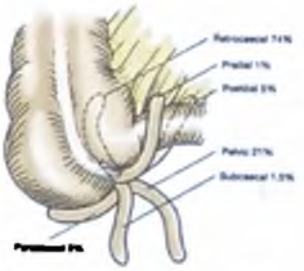
Positions: variable

1- Retrocaecal 74% 2- Pelvic 21% 3- Paracaecal 2%

4- Subcaecal 1.5% 5- Preileal 1% 6- Postileal 0.5%

7- Very rarely subhepatic in malrotation or left sided in *situs inversus totalis*

Surface anatomy: The base is the most fixed point, and is represented by Mc Burney's point at Junction of the lateral 1/3 and the medial 2/3 of a line extending from the umbilicus to the Ant. Sup. Iliac Spine (ASIS).



Various positions of the appendix

Blood Supply:

Arterial: appendicular art. from the ileocolic art. from sup. mes. art.

Venous: appendicular vein into the ileocolic V. into sup. mes. V.

Nerve supply and lymphatic drainage:

As the caecum

Applied Anatomy

The marginal artery provides a good anastomotic channel that is very helpful in anastomosis.

- The appendicular art. is endartery and runs parallel to the appendix in the mesoappendix so, it is usually included in the inflammatory process of appendicitis → thrombosis → gangrene.

The ileocaecal junction is guarded by one way valve (ileocaecal valve) which prevents the regurge of the colonic content into the ileum so, the small intestine doesn't show in the barium enema films except if the ileocaecal valve is incompetent (distal obstruction).

The ileum enters the caecum in acute angle called the ileocaecal angle. Widening of this angle predisposes to intussusception.

- The sup. mesenteric art. has a wider origin and more in line with abd. aorta than the inf. mes. artery therefore, embolus from the heart or aorta lodges more likely in superior mesenteric artery or its branches.

Resection anastomosis of colon carries risk of leakage due to :

1- High bacterial content of colon. 2- Continuous gaseous distention.

3- Incomplete serosa. 4- Weak musculosa. 5-Poor vascularity.

I-Congenital Diseases

1-Intestinal atresia

Aetiology:

Due to intrauterine vascular accident like volvulus or intussusception leading to aseptic necrosis and resorption of the affected segment.

Ileal atresia

Pathology: (that of int. obst.)

Sites: the ileum is the commonest site.

Clinical picture:

The condition starts shortly after birth with:

* Bilious vomiting * Abdominal distention. * failure to pass meconium

Investigations:

1-Stool analysis: absence of Lanugo hair. *2- Plain Xray(erect)*: multiple fluid level.

3-Barium enema: microcolon (unused colon).

Treatment:

Resection anastomosis of the atretic segment.

2-Intestinal Duplication

Aetiology: Vacculation disorder leading to double lumen, separate or communicating.

Complications - The accessory lumen may distend causing intestinal obstruction.

- Enterogenous cyst may be derived from the accessory lumen.

3-Mesenteric cysts

Aetiology & types:

1- Chylolymphatic cyst: congenitally sequestrated lymphatic tissue (has no efferent communication). It has thin wall and independent blood supply.

2- Enterogenous cyst: -It is a diverticulum from the mesentric border or derived from duplication of the intestine which becomes sequestrated from intestinal lumen.

-It has a thick wall & common bl. supply with the related loop

Mesenteric cyst

3- Dermoid and hydatid cyst.

4- False cysts: encysted haematoma, cold abscess, degenerating tumour or gas cyst.

Clinical picture:

Painless abdominal swelling showing the characteristic" *Tillaux's triad*"

1- Fluctuating swelling near the umbilicus.

2- There is restricted mobility in a plane along the root of mesentry but it is freely mobile in a plane perpendicular to it.

3- It is dull on percussion but with a zone of resonance around (the intestines) and a band of resonance across (the related loops of intestine).

Treatment:

Surgical removal by enucleation or resection anastomosis of the related loop of intestine.

4-Intestinal malrotation

Pathology:

Incomplete rotation of the midgut →caecum remains in the right upper quadrant and the duodenojejunal (DJ) junction to the right of vertebrae. This short mesenteric base (*between cecum & DJ)* leads to volvulous. Also, there are adhesive bands crossing the duodenum called Ladd's bands which cause duodenal obstruction..

Clinical picture:

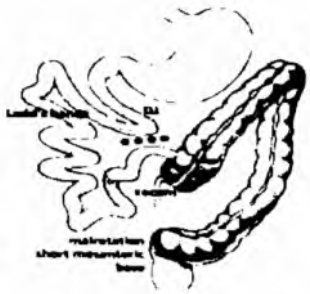
1-It may present at any age but usually in neonates.

2-Bilious vomiting with little or no abd. distension.

Investigations

1-Plain Xray erect: Double Bubble sign.

2-Barium enema: abnormal high position of the caecum.



Intestinal malrotation

Treatment:

Ladd's operation: Division of Ladd's bands and displacing caecum to the left, and appendectomy.

5-Volvulus neonatorum

Aetiology

Failure of absorption of mesentry of the ascending colon so the whole midgut hangs freely from a narrow mesentric attachment which predisposes to volvulus in a clockwise direction.

Clinical picture:

Few days (2-3) after birth there are:

-Biliary vomiting. - Little or no distension.

-Bloody stools and peritonitis are signs of strangulation.

Differential diagnosis:

- From other causes of neonatal intestinal obstruction especially intestinal atresia.

item	Congenital atresia	Volvulus neonatorum
Type of obstruction	Simple	Strangulated
Onset	Immediately or shortly after birth	2 -3 days after birth
Abd - distension	Develops gradually	No or little distension
Bloody stool	No	May be
Lanugo fair	Absent in meconium	Present

Treatment: urgent exploration

- Undo the twist of the whole midgut in anticlockwise direction with division of Ladd's bands.
- Inspection of the bowel:

  If still viable and healthy → return to the abdomen, after excision of Ladd's bands.

  If nonviable → resection anastomosis. (high mortality).

  If doubted viability → the loops in doubt are returned to the abdomen for second look operation 12-24 h later.

6-Meconium ileus

Aetiology: -

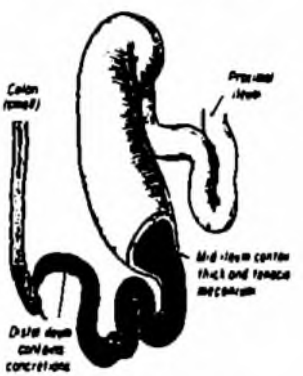
It occurs in about 20% of cases of " cystic fibrosis " which is an autosomal recessive hereditary disease affecting, pancreas, lung, liver, gut & skin.

Pathology (mucoviscidosis):

1-The defect lies in the exocrine glands and is characterized by production of viscid mucous and excess loss of Chloride in sweat.

2-The terminal ileum is obstructed by thick meconium

3- Progressive pancreatic fibrosis.



Meconium ileus

Clinical picture:

- Positive family history.
- Manifestation of neonatal intestinal obstruction:

  -Bilious vomiting -Distension. -Failure to pass meconium.

Investigations:

1- Plain X ray erect: *ground glass appearance* (mixed stools and gas).

2- Barium enema: small sized colon (microcolon). Reflux of barium into the narrow ileum showing filling defects due to pellets of meconium.

3- Estimation of chloride conc. in sweat : level over 60 mg /L is diagnostic

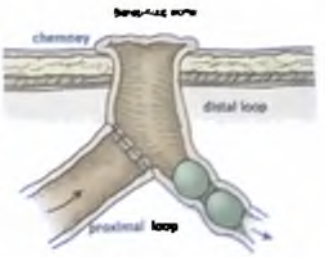
Treatment:

1- Conservative ttt:

N/G suction, I.V. fluids, gastrografin enema

II- Surgical ttt: (*Bishop Koop operation)*

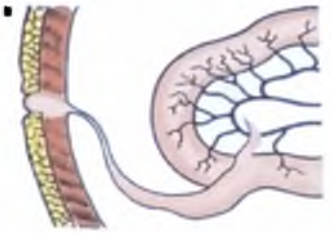
It entails resection of the dilated loops, end to side anastomosis and the end of distal loop is brought as end enterostomy This temporary enterostomy is used to guard against anastomotic rupture (chimney or decompressing fistula)



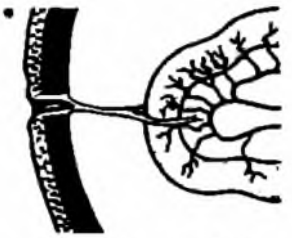
Bishop Koop operation

7-Anomalies of vitello-intestinal duct

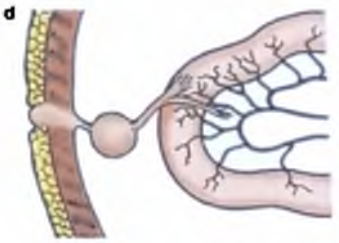
Cong.intestinal fistula



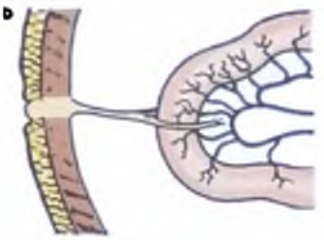
Meckel's diverticulum



Cong. Umbilical sinus



Enterocystoma



Fibrous band

1- *Persistence of the whole patent duct* → congenital umbilical. faecal fistula.

2- *Persistent patent intestinal end of the duct*→ *Meckel's diverticulum.*

3- *Persistent patent umbilical end of the duct* → umbilical sinus that becomes everted outwards by the intra abdominal pressure.

4- *Persistent patent middle part of the duct with obliteration of both ends* → enterocystoma or vitelline cyst behind the umbilicus.

5- *Persistence of the whole obliterated duct* → vitello-intestinal band which may strangulate loop or act as an axis for volvulus.

8-Meckel's diverticulum

Meckel's diverticulum

Aetiology:

Persistent patent proximal portion of the vitello-intestinal duct.

Pathology: Disease of two.

-It occurs in **2%** of population.

-It symptomatizes only in **2%** of them.

-It is about **2** inches long.

-It projects from the antimesentric border of the ileum about **2** feet from the ileocaecal junction.

-It contains heterotropic gastric and pancreatic tissues in **20%** of cases.

-It is sometimes attached to the umbilicus by a fibrous band at its apex, which represents the obliterated distal part of the vitello-intestinal duct.

Clinical picture & complications:

- Uncomplicated cases are asymptomatic and patients present with complications:

1-Bleeding is the commonest complication in children. It is due to peptic ulceration of adjacent ileal mucosa by acid secretion of heterotopic gastric mucosa. It leads to dyspepsia similar to that of D.U., but pain is felt around the umbilicus. Commonly, the patient bleeds per-rectum and rarely presents with perforation.

2--Meckel's diverticulitis: the condition is similar to acute appendicitis. It may proceed to perforation and peritonitis.

3-Intestinal obstruction: may develop because of:

i) Intussusception: the swollen base of the diverticulum act as the apex.

ii) Internal herniation: where the fibrous band attached to umbilicus strangulates a loop.

iii) Volvulus of the related loop of ileum around the fibrous band.

4-Littre's hernia: strangulation of the diverticulum in femoral or inguinal hernia without obvious intestinal obstruction.

Investigations:

1-Barium follow through: rarely, Meckel's appears in barium examination.

2- Pentagastrin-stimulated technetium-99m isotope scan:: to localize heterotopic gastric mucosa in diverticulum in case of bleeding per rectum.

Treatment:

-If it is encountered during barium studies →only give the patient a report about it.

-If it is encountered during abdominal operations for unrelated condition & the general condition of the patient permits, it should be resected to avoid the subsequent complications.

-If it is inflamed (Meckel's diverticulitis), resection is done:

*if the base & related loop of ileum are healthy, do tangential excision and suturing.

*If the base is grossly indurated with congested or inflamed loop of ileum → resection anastomosis of the related loop.

9-Hirschsprung's disease

(Congenital megacolon)

Aetiology: Swenson's theory

Due to congenital absence of parasympathetic ganglion cells in a segment of the distal bowel extending upwards from the anus, (the ganglion cells migrate caudally in the gut.).

Pathology:

Site:

- In 90 % of cases, the aganglionosis extends from the anus to the rectosigmoid. It may extend beyond the sigmoid and rarely, involves the whole colon (total aganglionosis).
- The aganglionic segment lacks normal peristalsis and contracts en masse in response to distension. This spastic segment produces functional obstruction that leads to hypertrophy and later dilatation of the proximal colon → megacolon.

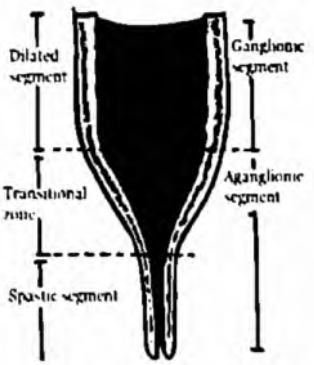
MAC:

There are three distinctive segments from proximal to distal:

1-*Dilated hypertrophied segment*: huge dilatation of the pelvic colon and may be proximal parts of the colon.

2-*Transitional zone* or cone (1-2 inches).

3-*Spastic segment*: including anal canal, rectum and may be, variable length of the colon.



Congenital megacolon

MIC:

Absence of ganglion cells of Auerbach's & Meissner's plexuses in both spastic and transitional segments, together with hypertrophied nerve fibres in the nerve plexuses.

Complications and causes of death:

1-Toxic absorption leads to delayed growth, and anaemia.

2-Recurrent attacks of enterocolitis.

3-Acute on top of chronic intestinal obstruction.

Incidence:

Common in first ♂ baby with positive family history.

Clinical picture:

The condition starts soon after birth with:

1-*Constipation*: The baby fails to pass meconium in the first 2 days. Only after insertion of a finger into the rectum then, the infant passes toothpaste like motions every few days.

2-*Abdominal distention*.

3-*Indentible faecal masses* may be felt in the iliac fossa.

4-*PR exam.* reveals free anus, empty rectum which grips firmly over the finger .

5-The patient commonly presents with complications e.g. enterocolitis.

Investigations:

1-*Barium enema*: hugely dilated colon with funnel shaped termination into a narrow spastic segment, which extends to the anal region.

2-*Anorectal manometry*: absence of the physiological reflex (relaxation of the anal sphincter in response to rectal distension or rectoanal reflex).

3-*Rectal biopsy*: reveals absence of ganglion cells.

Differential diagnosis:

1-From other causes of huge abdominal distension in infants *"spindle shaped infant"* :

- Wilm's tumor.
- Neuroblastoma.

2-From acquired "idiopathic "megacolon.

- It occurs due to faulty bowel habits, anal stenois or spasm, or abuse of purgatives.
- It occurs in older age group (usually childhood).
- PR exam. is painful, with spastic sphincter, and the rectum is full of inspissated faeces.
- Barium enema: shows dilatation of both colon and rectum down to the anal canal.
- Rectal biopsy and anorectal manometry are normal.
- Treatment is mainly conservative by sound bowel habits and training. Rarely in resistant cases with severe abdominal distension, one stage sigmoid colectomy is needed.

Treatment:

It is always surgical

I-Preoperative preparation through:

1 Improving the general condition of the patient.

2-Colonic lavage with saline enemata (but not with the tap water) to avoid water intoxication.

3-Preliminary colostomy is usually needed especially in old children.

II: Operations:

When the baby weighs 10 kg, one of the following operations is performed

1-Swenson's operation:

Combined abdominoperineal resection of the dilated and spastic segment with extracorporeal colo-anal anastomosis.

Disadvantages:

1-Extensive pelvic dissection with risk of injury to bladder & sex organs innervation (incontinence & impotence).

2-Loss of rectal sensation and storage capacity.

Swenson op. Step I

Swenson op. Step II

Swenson op. Step III

2-Duhamel's operation:

After resection of the dilated segment down to the peritoneal reflection, the rectal stump is closed and the proximal colon is anastomosed to its post. wall just above the sphincter. In modified Duhamel operation, the spur between the colon and rectal stump can be crushed or divided with stapler to have one pouch with preserved rectal sensation in its anterior wall and normal peristalsis in its posterior wall.

Disadvantages:

1-Faecal impaction in the blind rectal stump

2- Pelvic dissection though minimized yet it is still dangerous.

Duhamel's operation

3-Soave's operation : (most popular)

Dissection–excision of the rectal mucosa with pulling of the normal colon through the remaining rectal muscular cuff to which it is sutured above the pectineal line. Finally, we get a pouch with normal peristalsis in its inner coat & preserved rectal sensation in its outer coat.

Advantages over Duhamel's operation:

No pelvic dissection or blind pouch.

- It can be done through transanal approach (transanal pull-through mucosectomy operation). Also, laparoscopic assisted trans-anal pull-through is recently introduced.

Modified Duhamel's op.

4-Lynn's operation: *rectal myomectomy*

1 cm wide strip of the rectal muscle is resected up to the transitional zone. It is successful in cases with ultrashort Hirshsprung disease

Soave's operation

4- Transanal endorectal pullthrough: dissection of rectal mucosa (can be done completely via anal route. The normal colon is pulled down and anastomosed to anal mucosa.

5- Laparoscopy can be used in all procedures for dissection of colon or facilitating transanal pullthrough.

Transanal pullthrough

Inflammatory conditions

1-Acute appendicitis

Actiology:

- Causative organisms: usually mixed infection. The commonest organisms are :

  Aerobic: E. coli, and streptococcus faecalis.

  Anaerobic: anaerobic streptococcus, Cl. Welchii and Bacteroids.

- Routes of infection : either:

  -From the lumen, (the commonest).

  -Bl. borne infection. Very rarely, from upper respiratory tract infection.

- Predisposing factors:

  Stasis of appendicular contents as in chronic constipation.

  1-Obstructive appendicitis: (2/3of cases). It is caused by:

  Lumen: - faecolith, F.B, and parasites.

  Wall: - fibrosis, stricture and lymphoid hyperplasia.

  Outside: - adhesions, kinks or mass.

  2-Non obstructive appendicitis: (1/3 of cases)

  Stasis is caused by ch. constipation predisposed to by low residual diet.

Pathology: there are two types:

1-Obstructive appendicitis. It passes through the following stages:

a) *Acute catarrhal appendicitis*: due to obstruction, the appendix becomes distended with mucous and inflammatory reaction (mucocele of the appendix).

b) *Acute suppurative appendicitis*: leading to empyema of the appendix.

c) *Acute gangrenous appendicitis*: due to thrombosis of the vessels, increased intraluminal pressure occurs with ischaemia and gangrene distal to the site of obstruction. Perforation occurs usually at the site of obstruction and usually before the protective adhesions takes place leading to diffuse septic peritonitis.

2-Non obstructive appendicitis:

It passes through the same stages, but with the following differences:

1-The progress of events is much slower that gives time for protective adhesions to take place, so appendicular mass is a common result and even if perforation occurs, it causes a localized form of peritonitis.

2- Mucocele and empyema do not occur.

3-Gangrene and perforation are rare and if they occurred, the tip is the usual site.

Complications:-

1-Appendicular mass: - is formed of inflamed appendix, caecum and loop of the terminal ileum wrapped by omentum.

-It may resolve spontaneously within 3 weeks or may proceed to abscess or peritonitis.

2- Perforation may lead to either:

* *Generalized peritonitis.*

* *Localized peritonitis* e.g. appendicular abscess, pelvic or subphrenic abscess.

3-Portal pyaemia: due to septic thrombosis of the ileocolic vein, which results in multiple pyaemic abscesses in the liver.

4-Chronic appendicitis: resolution of acute inflammation with fibrosis (short white appendix) liable to recurrent attacks.

Incidence: -

It is the commonest cause of acute abdomen. It is common in:

- Adolescence and young adults, rare in children and elderly.
- Civilized communities consuming constipating low residual diet e.g. meat.
- Sedentary workers because they have tendency for constipation.

Clinical picture:

I- Non complicated cases

Symptoms:

1-Pain: -

Type: colicky pain in obstructive and dull aching in non-obstructive cases.

Site: first felt around the umbilicus (Both appendix & umbilicus are innervated by T10). Then shift to the right iliac fossa when the parietal peritoneum is inflamed.

Pain shift is diagnostic.

Increased by: coughing, movement and pressure & not improved by antispasmodics.

2-Nausea: more than vomiting and precedes pain.

3-Anorexia: is almost always present.

4-Constipation: is common, but diarrhea may occur.

5-*Tenesmus*: frequency and dysuria may occur with pelvic appendicitis.

Signs:

I-General:

Usually mild fever and tachycardia, high fever may indicate perforation.

II- local:

1-Localized tenderness in the right iliac fossa maximum over *Mc. Burney's point* (The junction of the medial 2/3 and the lat. 1/3 of a line extending form the umbilicus and the Rt. (ASIS).

2-Rebound tenderness & cough tenderness: Pain on releasing local pressure or on coughing indicates involvement of parietal peritoneum.

3-Rovsing sign: Increased pain in the right iliac fossa on pressure over the left iliac fossa due to displacement of the abdominal viscera towards the inflamed appendix.

4-Hyperesthesia over Sherren's triangle:

The triangle is bounded by the umbilicus and the two ends of the inguinal ligament

5-Muscle guarding and rigidity are often late and denote impending perforation.

6-Special signs: according to the site of the appendix.

a -In retrocaecal appendicitis:

**Psoas sign:* hyperextension or flexion against resistance of the hip joint is painful. Sometimes, psoas spasm is sufficient to cause flexion of the hip.

*Ureteric irritation may give pain similar to ureteric colic.

b - In pelvic appendicitis:

*Tenderness may be absent over the Rt. iliac fossa and elicited only on PR. or PV. exam.

**Zachary Cope sign*: Flexion and internal rotation of the hip may cause pain in the suprapubic region if the appendix is related to obturator internus muscle.

c - In subhepatic appendicitis:

*The pain and tenderness are in the right hypochondrium (DD. acute cholecystitis).

II - Complicated cases

Patients often present with complications.

1-Appendicular mass:

*There is recent history of acute appendicitis with constitutional manifestations.

*Abdominal examination reveals firm tender mass in the right iliac fossa with ill-defined margin and overlying muscle guarding.

2-Appendicular abscess:

*The pain becomes severe and throbbing & the fever becomes high and hectic.

*Symptoms of pelvic organs irritation are usually marked (tenesmus and frequency).

3-Acute diffuse peritonitis:

The picture does not vary form the general picture of generalized peritonitis.

4-Portal pyaemia:

High fever, rigor, mild jaundice and tender hepatomegaly are the main manifestations.

5-Chronic (recurrent) appendicitis:

Usually, there is a history of recurrent subacute attacks. There are anorexia, dyspepsia, nausea and intermittent dull aching pain in the right iliac fossa with mild tenderness.

Investigations:

1-Total and differential leucocytic count: usually there is mild polymorphnuclear leucocytosis.

2-Plain X-ray abdomen in erect position: to exclude lower ureteric stone and gas under diaphragm (Q. perforated peptic ulcer) .Perforated appendicitis very rarely gives free intraperitoneal gas, as it is usually obstructive.

3-Abd U/S: very helpful in diagnosis of appendicular abscess or mass and diffuse or localized peritonitis. In addition, it is important to exclude other causes e.g. tubo ovarian causes.

Differential diagnosis:

I-Form other causes of acute abdomen (see later).

II-Appendicular mass should be differentiated from masses in the Rt. iliac fossa (see later).

Treatment:

I- Uncomplicated cases:

Appendicectomy either open or laparoscopic should be done to avoid complications.

Urgent operation is indicated in the following conditions:-

1- Children: due to short under-developed omentum.

2- Elderly patients: as atherosclerosis predispose to early gangrene and perforation.

3- Pregnancy: As the omentum is raised up by the growing uterus and to protect both the mother and baby, form the hazard of abortion or premature labour.

4- Diabetic and immunocompromized patients or those under corticosteroid therapy due to low resistance and decreased tendency for localization.

5- Patients who had taken potent analgesic e.g. narcotics as they may mask serious complications as perforation.

II- Complicated cases.

A- *Cases with appendicular mass*: →conservative ttt = *Ochsner Sherren's regimen* because operation is difficult and may precipitate faecal fistula or spreading peritonitis.

It consists of:

1-*Rest in semi-sitting or modified Fowler position* to rest the abdominal wall & if collection occurs, it would be in the pelvis (not in the subphrenic space) with milder toxaemia.

2-*NPO & I.V. Fluids for 48 h.* to rest the GIT.

3-*Broad spectrum antibiotics.* 4-*Antispasmodics.*

5- *Local hot fomentations or poultice application* (counter-irritant & increases vascularity).

6-*Observation:* is the cornerstone of conservative ttt.

- General: for toxaemia, fever, & pulse.
- Local: for size of mass, tenderness, guarding and rigidity.
- Laboratory through daily leucocytic count and imaging through abdominal U/S.
- Observations should be recorded in a chart.

* If the general condition of the patient improves, conservative ttt is continued until complete disappearance of the mass, and then appendicectomy performed 3 months later.

* If the condition worsens with rising pulse rate and fever, increasing pain, tenderness and or appearance of signs of generalized peritonitis or portal pyaemia → stop conservative ttt and prepare for urgent operation.

B- Cases with appendicular abscess: extraperitoneal drainage & appendicectomy 3 months later (interval appendicectomy).

C- Cases with generalized peritonitis: Resuscitation → exploration → appendicectomy + peritoneal lavage + drains.

D- Cases with portal pyaemia: urgent appendicectomy with ligation of the ileocolic vein under cover of broad-spectrum antibiotics.

E- Cases with recurrent appendicitis: appendicectomy.

Appendicectomy

Steps:

- *Incision*: *Mc Burney's incision* (2 inches long, oblique gridiron incision perpendicular to a line joining the umbilicus and Rt. ASIS and centered over Mc Burney's point).
- The ext. oblique aponeurosis is split along the direction of its fibres, and the internal oblique and transversus abdominis muscles are also split along their fibres i.e. perpendicular to that of the ext. oblique (gridiron incision).

- The peritoneum is opened, the caecum is delivered into the wound and appendix is identified.
- The mesoappendix is ligated & divided and the base of appendix is crushed, ligated, & divided. The stump may then be inverted in the caecum with purse string sutures.
- The wound is closed in layers without drains, except if there is peritoneal contamination.

Complications of appendicectomy:

1- Haemorrhage: either intraperitoneal due slipped ligature of mesoappendix, or in the lumen of the colon from the inverted stump revealed as bleeding per rectum.

2- Infection: either wound infection or intraperitoneal sepsis e.g. iliac or sub phrenic abscess.

3- Faecal fistula may develop due to one of the following:

- imperfect ligation of the base.
- inadvertent injury to the caecum or the ileum during operation.
- Specific pathology e.g. TB. or Crohn's disease.

4- Incisional hernia usually after wound infection.

5- Direct inguinal hernia due to injury of the ilioinguinal nerve.

Differential diagnosis of mass in the Rt. iliac fossa.

I-Masses coming from the contents of the fossa:

1-Appendicular mass (commonest). 2-Cancer caecum (next common).	5- Aneurysm of the ext. iliac art. 6- Tumours in undescended testicle.
3-Ileocaecal tuberculosis. 4- Crohn's disease.	7-Iliac lymphadenitis. 8- Iliopsoas abscess.

II- Masses coming from above (abdomen):

1-Liver e.g. huge hepatomegaly. 2- Gall bladder e.g. mucocele.

3- Spleen e.g. huge B. splenomegaly. 4- Kidney e. g mobile or ectopic.

III- Masses coming from below (pelvis):

1- Ovaries e.g. ovarian cyst or tumour. 2- Tube e.g. tubo ovarian mass.

3- Uterus e.g. fibroid.

IV- Masses coming from front (abd. wall):

Lesions of the ant. abd wall (lipoma, fibroma, sarcoma,& interstitial hernia) .

V- Masses coming from behind (retroperitoneal):

1- Retroperitoneal tumours. 2- Hip bone: osteomyelitis or tumour.

NB. Mass in the Lt. iliac fossa has the same possibilities with the following differences

I- Masses from the contents:

1-B. pericolic mass (commonest in Egypt). 3-Acute diverticulitis.

2-Cancer sigmoid (faecal impaction). 4-Amoeboma (amoebic pericolic mass).

5, 6, 7, 8, as in right side.

II- Masses coming from above:

1- Splenomegaly (ch. myeloid leukemia). 2- Ectopic kidney (very rare)

III- Mass coming from below, front & behind as the right side.

2-Intestinal tuberculosis

Actiology: Intestinal infection with T.B bacilli.

Causative organism: Mycobacterium tuberculosis, which is an acid fast, alcohol fast bacillus.

Routes of infection: there are two types of oral infection, which cause two pathological types:

1-Secondary infection: the commonest, due to swallowing of infected sputum in patients with pulmonary T.B (Human T.B bacilli). It causes ulcerative T.B. enteritis.

2-Primary infection: due to swallowing of infected food [e.g. milk (usually the bovine T.B bacilli] causing hyperplastic ileocaecal T.B.

Pathology:

1-Ulcerative type: -

-*Site*: terminal ileum (why?) due to its high absorptive power, relatively stagnant contents and abundant lymphatics. The bacilli reach the lymphoid follicles, and due to the high virulence of the organism and the low resistance of the patient, sloughing occurs.

MAC: -*The affected segment* is swollen, congested & the serosa shows multiple tubercles.

-*The mesentry* shows enlarged LNs which may caseate.

-*The mucosa* shows inflammation, sloughing and formation of multiple transverse ulcers with pale granulating floor and undermined edges.

2- Hyperplastic type:

Site: the ileocaecal region. The bacilli reach the lymphoid follicles, but due to the relatively high resistance of the body, good local reaction with hyperplasia & induration occurs.

MAC: -*The affected segment* becomes greatly thickened and indurated with narrowing of the lumen and shortening of the ascending colon.

-The mesentry shows enlarged LNs without caseation.

-*The mucosa* shows inflammation.

Complications:

I-General:

1-*T.B. toxaemia.* 2-*Anaemia and cachexia.*

II-Local:

1-*Intestinal obstruction*: very common due to healing of the transverse ulcer with fibrosis and stricture. This leads to narrowing of the lumen and serosal adhesions.

2-*Perforation*: rare due to fibrosis and thickening.

3-*Abscess and fistula formation:* (rare) Internal or external fistulae may develop.

4-*Malabsorption* due to chronic diarrhea, stagnant loop syndrome & diseased ileum.

Clinical picture:

1-Ulcerative TB. enteritis:

-Evidence of pulmonary tuberculosis is usually present with T.B toxaemia.

-Abdominal colic.

-Chronic bloody diarrhea.

- Picture of complications: anaemia, & loss of weight.

2-Hyperplastic ileocaecal T.B.:

-Attacks of abdominal colic with subacute or acute on top of chronic intestinal obstruction.

-Intermittent diarrhea (steatorrhea).

-Mass in the right iliac fossa (firm and tender).

-Picture of complications.

Investigations:

1-Laboratory: - Blood picture. ESR, sputum analysis and tuberculin test.

2-Radiological:

-Chest X ray to detect pulmonary TB.

-Plain X ray abdomen: may detect calcified mesentric LNs.

-Barium follow through, may show one or more of the following signs:

- Rapid evacuation of barium from the lower ileum due to hypermotility →failure to show barium in the terminal ileum, caecum and most of the ascending colon (*barium jump*).
- High position of the caecum (subhepatic) with widening of the ileocaecal angle.

3-Peritoneal biopsy through laparoscopy or laparotomy is the surest method.

Treatment:

Rt. hemicolectomy

I-Medical treatment: (All cases should receive anti TB drugs)

1- Sanatorium admission.
2- Good nutrition & improve the general condition.
3- Antituberculous drugs e.g. rifampicin, I.H, and Pytizinamide.

II- Surgical treatment:

Indications:

1-Development of complications e.g. obstruction, perf. & fistulae.

2-Hyperplastic type because differentiation from cancer is difficult.

Procedure:

-Resection of the diseased segment with primary anastomosis e.g. Rt. Hemicolectomy and ileotransverse anastomosis

- Ileotransverse colostomy is done if adhesions are extensive.

Ileotransverse colostomy

3-Crohn's disease

(Regional enteritis)

Definition

It is a chronic cicatrizing non-caseating granulomatus inflammation of the bowel.

Aetiology:

unknown, but may be due to abnormal cell-meditated immune reaction to ingested antigens

Pathology:

Site: -It may affect any part of the alimentary tract from the mouth to the anus but, the commonest site is ileocaecal region (80%) ,followed by colorectal region, gastroduodenal region and the oesophagus in that order hence, the name *regional enteritis*.

-It usually affects multiple segments in the GIT with healthy segments in between and hence, the term skip *lesions*.

MAC: -*The affected segment* is thickened, narrowed (stricture), and shows characteristic creeping of the mesenteric fat to the serosal surface of the bowel *fat wrapping*.

-The mesentry is thickened & contains enlarged LNs, which neither caseate nor calcify.

-The mucosa is fissured with deep ulcers, which may surround islands of intact mucosa over oedematous submucosa giving *cobble stone appearance.*

MIC: - Early: There is non-caseating granuloma including lymphocytes, plasma cells and giant cells infiltrating all layers of the affected segment

- Late: There is dense fibrosis involving all layers of the bowel leading to stricture

Complications:

I- General complications:

-Weight loss, anaemia and hypoprotienaemia.

II-Local complications:

1-Intestinal obstruction, due to narrowing of the lumen and serosal adhesions.

2-Stagnant loop syndrome → steatorrhea → malnutrition & vitamins deficiency.

3-Perforation: which is slow and preceded by adhesions leading to abscesses & fistulae.

4-Fistulae: may be internal (e.g. entero-enteric & entero-vesical) or external (e.g. entero-cutaneous or perianal).

5- Malignant transformation is not common.

Incidence:

It is rare in Egypt.

It is common in young adults of both sexes.

Clinical picture

I Acute Crohn's disease: (5% of cases)

-It simulates symptoms and signs of acute appendicitis.

II - Chronic Crohn's disease: with different modes of presentations:

1-*Pain*: The commonest symptom.

-It is due to partial (subacute) obstruction.

Character :colicky. *Intensity* : mild to severe.

Site : paraumbilical or lower abdominal. *Radiation*: localized.

Precipitated by: meals. *Relieved by* : defecation.

2-*Diarrhea* (>90% of cases)

- It is due to stagnant loop syndrome and steatorrhea.

-It is continuous or intermittent with passage of loose stool.

3- *Mass* in the Rt. iliac fossa, which is firm & tender.

4-*Complications*: may be the first presentation

Investigations:

1-Barium follow through and enema may show:

-Narrowing of the terminal ileum producing (*string sign of Kantor*).

-cobble stone pattern after evacuation of barium.

-Complications e.g. internal or external fistulae.

string sign of Kantor

Differential diagnosis:

1-Acute Crohn's disease is very difficult to be differentiated from acute appendicitis.

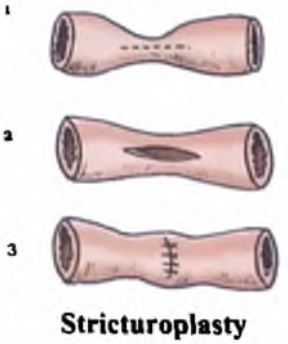
2-Chronic Crohn's should be differentiated from TB., carcinoma, and ulcerative colitis.

Treatment

<u>I-Conservative treatment:</u>

For all cases .It runs on the same lines as for ulcerative colitis.

<u>II Surgical treatment.</u>



Stricturoplasty

Indications:

1-Failure of medical treatment (no response or side effects).

2-Complications e.g. obstruction, abscess or fistulae.

Procedures: "*Least procedure and minimal resection*"

-*Resection anastomosis*: e.g. Rt. hemicolectomy with ileo-transverse anastomosis.

- *Bypass*: ileotransverse colostomy.

-Stricturoplasty: longitudinal incision through the stricture which is closed transversely

4-Ulcerative colitis (Proctocolitis)

Definition:

It is nonspecific ulcerative disease affecting the mucosa and submucosa of the distal bowel.

Aetiology: unknown → theories:

1-Infection: no certain organism could be incriminated.

2-Psychosomatic disorders: exacerbation by emotions.

3-Allergic: allergy to milk protein was suggested.

4-Familial: due to increased familial tendency, which suggest genetic predisposition.

5-Autoimmune disease: circulating antibodies against colonic cells cytoplasm were detected in the sera of most patients.

Pathology:

Site: the rectum is affected in about 95% of cases. The disease spreads proximally to the colon, and <u>*backwash ileitis*</u> occurs in 10% of cases.

MAC: -*The mucosa* shows multiple minute ulcerations, which coalesce to form larger ulcers. The mucosa in-between them become oedematous & hypertrophied to form <u>*pseudo-polyps.*</u>

-*The affected bowel* is shortened due to muscle spasm, and later, due to fibrosis.

-*The mesentry* is thin and free.

MIC: Early, there is inflammatory cells, mainly neutrophils, infiltrating the crypts of Liebrkhun to form crypt abscesses which burst later forming multiple minute ulcers.

Complications:

I- General complications:-

1-Toxaemia, malnutrition, loss of weight, anaemia and vitamin deficiency.

2-There is high incidence of erythema nodosum, pyoderma gangrenosa, ankylosing spondylitis & polyarthritis, Uveitis, iritis and conjunctivitis & secondary sclerosing cholangitis.

II- Local complications:

1-*Massive bleeding*: the second commonest cause of massive colonic hage .

2-*Intestinal obstruction*: is rare due to stricture at the rectosigmoid junction.

3-*Perforation*: usually leads to generalized peritonitis.

4-*Abscesses and fistulae*: pericolic or ischiorectal abscesses, recto- vaginal or perianal fistulae.

5-*Toxic megacolon:* in the acute fulminant type.

6-*Malignant change*: especially in chronic continuous type.

Incidence:

- The disease is rare in Egypt. but common in western countries and Jews.
- It is more common in young adult females.

Clinical picture:

I-Acute fulminant type (5% of cases):

-Toxaemia and fever.

-Lower abdominal colics, severe diarrhea containing mucus, bl. and pus with severe tenesmus.

-Marked abdominal distention if toxic megacolon develops.

-Rapid dehydration and shock with mortality rate about 20%.

II Chronic type (95% of cases):

A-Chronic intermittent type:

-The disease comes in attacks of moderate severity separated by free intervals of varying length.

-Attacks (exacerbations) are evoked by Stress (emotional or physical) Sepsis, Diet (e.g. milk products), or Drugs (e.g. laxatives & antibiotics).

-The main symptoms are chronic bloody diarrhea, abdominal colic & weight loss.

-Abd. exam. -Tender palpable colon especially over the Lt. iliac fossa .

-PR. exam: fissured, tender and spastic anus. Blood & pus over the gloves.

-Picture of other complications may be detected.

B - Chronic continuous (remittent) type:

The patient is not completely free in the intervals. Symptoms persist but they vary in severity

Investigations:

1-Stool analysis: to exclude other causes of colitis (B, amoebic & bacillary).

2-Barium enema: not in the acute stage (exacerbations), as it may predisposes to perforation or toxic megacolon. During remissions, it shows narrow short colon with loss of haustrations. (*Ribbon shaped colon*) or lead pipe appearance.

Lead pipe appearance

3-Colonoscopy: It may detect ulcers or pseudo-polypi.

4-Rectal biopsy: It is very valuable to:

1- Establish the diagnosis

2- Detect the effect of therapy.

3- Exclude cancer

Differential diagnosis:

-From other common causes of colitis (amoebic, bilharzial, bacillary, salmonellosis ... etc)

Treatment:

I-Conservative treatment: It gives good results in 80% of cases.

1-Rest: both mental and physical rest.

2-Diet: -low residue, high protein diet with minerals and vitamins supplementation.

-Milk, cellulose, fat, spirits, spices, smoking all should be restricted.

3-Drugs:

1- Salazopyrine (Sulphaphthalidine + Salicylates): Sulphathalidine is antiseptic & salicylic acid is anti-inflammatory.

2- Corticosteroids: may induce remission

3- Antidiarrheal drugs:

II - Surgical treatment:

Indications:

1- Failure of medical treatment for both acute and chronic types.

2- Development of complications: massive haemorrhage, perforati

Ileal pouch –anal

Procedures:

1-The procedure of choice nowadays is restorative coloproctectomy with ileal pouch (J, S or W shaped) anal anastomosis.

2-If the rectum is spared (5% of cases), some advocate total colectomy and ileo-rectal anastomosis with periodic observation of the rectal stump.

3- Ileostomy alone may be performed in complicated cases with bad general condition.

5-Bilharzial colitis

Actiology:

- Causative organism:

Bilharziasis or schistosomiasis is caused by the parasitic worm schistosoma. Intestinal bilharziasis is caused by schistosoma mansoni (85%) and schistosoma haematobium (15%).

- Route of infection: = life cycle:

- Man is infected by contact with water infected with cercariae (infective stage) which penetrate the skin actively leaving their tails out.
- Cercariae reach the veins to the right side of the heart to the lung passing through the capillaries to the left side of the heart to the general circulation.
- Only those who reach the hepatic and mesentric circulation will grow to adult stage.
- Adult male worms carry females and swim against stream in the portal vein then in the inf. mesentric vein selectively (due to relatively slow stream) to reach the pelvic and mesentric plexus where they lay eggs that penetrate the wall of the colon and rectum to pass in stool.
- If eggs reach fresh water, they hatch to liberate miracidia, which swim searching for their appropriate snail vector (Biomphlaria alexandirina) where they multiply and grow into cercariae that are discharged from the snail into water where they swim searching for man.

Pathology:-

Bilharzial lesions are due to allergic reaction to an antigen in the ova. The severity of the disease depends upon the number of ova.

Site: The rectum and pelvic colon are the parts, which suffer much more, as they are common sites for ovideposition.

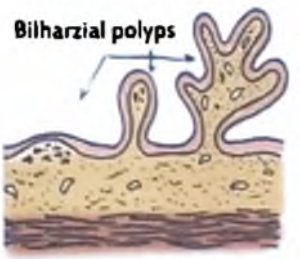
Macroscopically: the disease occurs in two forms:

1-The submucous form: The inflammation is localized to the submucosa. The mucosa is hyperemic and shows:

a- *Sandy patches*: small dirty yellow raised patches. It is due to atrophy of mucosa over the submucosal B. granuloma.

b- *Ulcers*: due to necrosis of mucosa or falling of polyps.

c-*Polyps:* due to raising of the mucosa by growing B. granulation tissue



2-Diffuse form: all coats are affected:

a- Mucosa: sandy patches, ulcers and papilloma .

b- Submucosa: thickening and fibrosis.

c- Musculosa: hypertrophied and infiltrated.

d- Serosa, appendices epiploicae, mesocolon: - hypertrophied and thickened forming a firm mass (B pericolic mass) simulating carcinoma.

Complications:

I- General complications:

1-Anaemia & loss of weight.

2-Liver: the backwashed ova cause periportal fibrosis and portal hypertension.

II-Local complications:

1-Bleeding per rectum.

2-Intestinal obstruction is extremely rare (the lumen is not encroached upon).

3-Perforation: is very rare and if it occurred, usually leads to pericolic abscess.

4-Rectal prolapse: due to straining and pulling down by polyps.

5-Carcinoma: may occur as a coincidental lesion but not due to bilharziasis.

Incidence:

-It was common in Egypt especially in the Lower Egypt as Biomphlaria Alexandrina is a flat snail that floats on the surface and reaches there. Now, it almost disappeared from Egypt.

-It commonly affects young adult male farmers in the countryside.

Clinical picture:-

- History: of bilharziasis or contact with infected water.
- Symptoms:

-General weakness, easy fatigability, and pallor.

-Tenesmus with bleeding per rectum (bilharzial dysentery).

-Symptoms of urinary bilharziasis are usually present.

-Complications may be the first presentation.

- Signs:

-Hepato-splenomegaly and other manifestations of portal hypertension.

-Mass in the Lt. iliac fossa, which is

* Tender. *Tubular in shape. *Nodular surface.

* Firm in consistency. *lying obliquely in the Lt. iliac fossa. *early mobile &late fixed

-P R exam.: may reveal rectal polypi.

Investigations:

1-Stool and urine analysis: RBCs, B. ova →Hatching test.

2-Serological tests for B.: ELISA, CFT. & Circumoval precipitation test (COPT.).

3-Barium enema: It shows "*honey comb appearance*" due to polypi which appear as multiple globular irregular filling defects of variable size in the rectum and pelvic colon.

4-Sigmoidoscopy: ulcers, polyps → rectal scrapping or biopsy.

Differential diagnosis:-

-Form other causes of colitis (dysentery): amoebic, bacillary.....etc.

-Bilharzial pericolic mass should be differentiated from other masses in the Lt. iliac fossa.

Treatment:

I-Medical treatment: (all cases should receive Anti B. drugs):

1- Anti B drugs: Praziquantel is the drug of choice.

2-Management of the liver condition, portal hypertension and other complications.

II- Interventional treatment: Endoscopic fulguration of B. polypi if small in number.

III Surgical treatment:

Indications:

1-Large number of polypi.

2-Bilharzial pericolic mass if differentiatiation from carcinoma is difficult.

3-Development of complications, which need surgery e.g. obstruction or perforation

Procedures:

- One stage partial colectomy with end-to-end anastomosis after preparation of the colon.

7-Typhoid enteritis

Actiology:

Causative organism: Salmonella typhi. Route of infection: Oral.

Pathology:-

Site: - The commonest site is ileocaecal region.

-The organisms invade the Payer's patches and pass through lymphatics to LNs and then to blood (bacteraemia) to different organs of the body.

-The pathology in the intestine passes through the following stages:

1-Oedema and hyperaemia of Payer's patches (1st week).

2-Necrosis of the overlying mucosa (2nd week) due to septic thrombosis of the vessels.

3-Ulceration of mucosa (3rd week) due to separation of sloughs. The ulcers are oval with their long axis in the long axis of the intestine (direction of Payer's patches).

Serious complications e.g. bleeding and perforation may occur in this stage.

4-Healing with minimal fibrosis and without strictures as the ulcers are longitudinal.

Surgical complications of typhoid

1-Paralytic ileus: is the commonest complication.

2-Intestinal bleeding: (in the 3 rd week).

-It manifests by abdominal colic & bleeding per rectum.

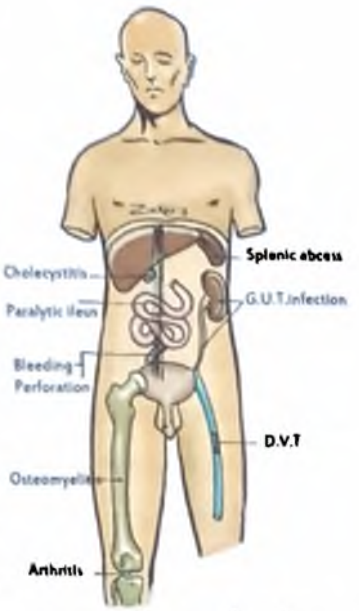
-It may be the 1st presentation (ambulatory typhoid).

3-Perforation may occur in the 3rd week.

-Perforation usually occurs in terminal ileum and may be multiple.

-It simulates the classic picture of generalized peritonitis but, with the following characteristic features:

- History of prolonged fever.
- The abdominal wall appears oedematous.
- Abdominal rigidity may be minimal due to toxaemia.
- The leucocytic count is not markedly raised or even reduced.



Surgical complications of typhoid

- X ray shows gas under diaphragm.

Treatment

Preoperative preparation:

-nasogastric suction *-I.V fluids-* *- Blood transfusion.*

-Antibiotics: if perforated typhoid ulcer is suspected, chloramphenicol (large doses) is given.

Operative ttt:

Urgent exploration with sealing of perforation, peritoneal lavage and drainage. If multiple perforations in a short segment, do resection anastomosis of the affected segment.

Prognosis: the mortality rate is 30%.

4-Cholecystitis: both acute and ch. cholecystitis and predispose to gall stones.

5-Splenic abscess.

6-Phlebitis: thrombophlebitis of the left common iliac vein (DVT.).

7-Osteomylitis.

8-Arthritis: all degrees of arthritis.

9-Genitourinary tract infection: typhoid pyelitis, cystitis and epididymo orchitis.

10-Zinker's degeneration of the rectus abdominis: hyaline degeneration may lead to rupture.

Clinical picture, Investigations & treatment: see medicine.

Diverticular disease

Definition:

Diverticulum is an abnormal pouch protruding from the wall of a hollow organ.

Aetiology:

I-Congenital:

It has the all three layers of the gut.

II –Acquired-

1-Pulsion diverticulum: -It occurs due to muscular incoordination leading to increased transluminal pressure → herniation of the mucosa through weak points in musculosa e.g. at sites of entrance of bl. vessels.

-Pulsion diverticula lack the muscular coat and so, they cannot evacuate themselves → stasis → infection → complications.

2-Traction diverticulum: It occurs due to traction on the gut wall by a nearby fibrosis e.g. T.B lymphadenitis.

3-Pseudo diverticula: They appear in contrast radiography. They result from epithelialization of ulcer crater or abscess cavity.

Pathology:

Site: It may arise in any part of the gut from the pharynx to the rectum.

-*Pharynx*: Zenker's diverticulum = pharyngeal pouch.

-*Oesophagus:* pulsion, traction and congenital diverticulum.

-*Stomach:* gastric diverticula.

-*Duodenum*: duodenal diverticula. It is common in the 2nd part at the entrance of common bile duct. It may compress it causing obstructive jaundice.

-*Jejunum & ileum:* Jejunoileal *diverticula* are rare expect Meckel's diverticulum, which is the commonest diverticulum. They are pulsion diverticula and are usually asymptomatic. They may lead to bleeding, perforation, obstruction (intussusception) or if multiple, may cause blind loop syndrome and malabsorption.

-*Caecum:* caecal *diverticulum:* is congenital (true) diverticulum, which is usually solitary. It may be inflamed giving the same picture of acute and chronic appendicitis.

-*Colon:* colonic *diverticula* = Diverticulosis coli (see below).

-*Rectum:* It is extremely rare.

Diverticulosis coli

Aetiology: acquired pulsion diverticula.

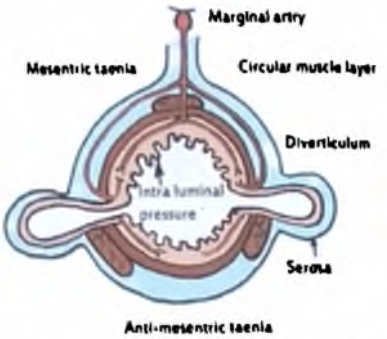
Muscular incoordination → increased intra colonic pressure → herniation of the mucosa through weak points at entrance of bl. vessels.

Predisposing factors: Chronic constipation and low residual diet.

Pathology:

Site: -the commonest site is the sigmoid colon (narrow).

-Diverticula occur in rows along the mesentric border of the antimesentric taenia.



Cross section of the colon

Complications:

1-Inflammation: - the commonest complication due to lack of the muscular coat →stasis → inspissation → obstruction → infection → diverticulitis.

2-Perforation which leads to peritonitis, either localized (abscess) or generalized.

3-Fistula: either external →colocutaneous (faecal) fistula or internal to nearby organs →. colovesical, colovaginal or coloenteric fistulae.

4-Intestinal obstruction: usually due to recurrent inflammation with fibrosis and stricture.

5-Haemorrhage: the close relation of the neck of diverticula to the long marginal artery predisposes to bleeding which is characteristically severe.

Diverticulosis coli is the commonest cause of profuse bleeding per rectum.

I-Uncomplicated cases (Diverticulosis coli)

Clinical picture:

-Asymptomatic but, some cases complains of "*irritable colon syndrome* ".

-Distension, heaviness and colicky pain in the left iliac fossa.

-Thickened tender colon on examination.

Investigations:

-*Barium enema*: may show narrow lumen + multiple globular shadows = barium filled diverticula or *Saw tooth appearance* due to segmental spasm → serrations.

- Colonoscopy: It may show the orifices of the diverticula

Saw teeth appearance

Treatment:

-Avoid or stop strong purgatives.

-High residual diet e.g. whole meal of bread, vegetables and fruits.

- Anti spasmodic and sedative for IBS

Endoscopic picture

II-Complicated cases

1-Diverticulitis:

Clinical picture:

- Appendicitis like symptoms but, the pain shifts to the left iliac fossa (DD. left sided appendicitis).
- Fever, tenderness and rigidity in the left iliac fossa.

Investigations:

Barium enema and sigmoidoscopy but are contraindicated during the acute attack as they may precipitate perforation.

Treatment:

I-Conservative: as Ochsner Sherren regimen (see before).

II -Surgical ttt is indicated in recurrent acute attacks. primary resection anastomosis of the affected segment is done after chemical and mechanical preparation of the colon.

2-Perforation & Peritonitis:-

Clinical picture:

-Either localized (pericolic abscess) or generalized faecal peritonitis.

-Symptoms and signs of acute peritonitis with severe toxaemia, due to high bacterial content.

Investigations: -

1-Plain x-ray erect: → gas under diaphragm.

2-Leucocytosis.

Treatment: surgical

Resuscitation + urgent operation.

1-Three stage procedure:

- *Stage I:* Proximal transverse colostomy and closure of the perforation(s).
- *Stage II:* Resection anastomosis of the affected segment after chemical and mechanical preparation of the colon (3 weeks later).
- *Stage III:* closure of the colostomy (2 weeks later).

Stage I
Colostomy + sealing

Stage II
Resection-anastomosis

Stage III
Closure of colostomy

2-Two stage procedure:

-*Stage I:* resection of the affected segment: and the proximal end is brought out as terminal colostomy and the distal end is closed as in Hartman's operation.

-*Stage II:* end-to-end anastomosis to restore the continuity.

Stage I:

Stage II

Two stage colectomy

On table lavage

One stage colectomy

3-One stage procedure:

-Resection of the affected segment with on-table colonic irrigation with copious amount of saline injected through a tube introduced through small stab in the terminal ileum or the base of the appendix till the draining fluid comes clear from the distal end of the colon then re-anastomosis in the same sitting.

3-Intestinal obstruction: -

Clinical picture:

-Symptoms of chronic obstruction (progressive constipation, abdominal distention. Sometimes, acute obstruction supervenes.

Investigations: Barium enema and Sigmoidoscopy to rule out malignancy.

Treatment: as in perforation.

4-Fistula: -

Clinical picture:

-External fistula usually follows drainage of pericolic abscess.

-Vesico colic fistula usually presents with pneumaturia, dysuria or even faecaluria.

Investigations: Barium enema, cystoscopy, & cystography are helpful.

Treatment: Fistulectomy & primary resection anastomosis of the affected loop of colon after colon preparation.

5-Haemorrhage: -

Clinical picture:

-Characteristically profuse and per rectum.

Investigations: -

-Selective angiography.

-Barium enema and colonoscopy have no role in massive colonic bleeding.

Treatment: -

-Resuscitation with bl. transfusion.

-Therapeutic selective embolization of the bleeding vessel.

-Total colectomy in persistent massive colonic bleeding with ileo- rectal anastomosis.

Causes of subphrenic gas (Gas under the diaphragm)

1-Postoperative: The commonest cause:

-Post-laparotomy. -Post-laparoscopy. -Post tubal insufflation.

2-Perforated peptic ulcer (the 2nd commonest cause).

3-Perforated typhoid ulcer.

4-Perforated colonic diverticulitis.

5-Rupture gut in blunt abdominal trauma.

6-Perforated appendicitis (rarely) why?

7-Peritonitis or subphrenic abscess by gas forming organisms.

III-Neoplasms

Tumours of small intestines

A) Benign tumours.

The commonest are adenoma, leiomyoma, lipoma, haemangioma, fibroma and neurofibroma.

They present with complications e.g. intestinal obstruction (intussusception) or bleeding.

B) Malignant tumours:

They are uncommon. They constitute only 2% of malignant neoplasms of GIT.

The four commonest types are adenocarcinoma, carcinoid, intestinal lymphoma, & lieomyosarcoma in descending order of frequency.

Carcinoid tumour

Argentaffenoma

Pathology:

Cells of origin: Argentaffin cells or enterochromaffin cells in the crypts of Lieberkhun.

Sites: Commonest site is the appendix, followed by ileum then rectum .Other rare sites include bronchus and gonads.

Clinical picture:

- Carcinoid of the appendix usually present as acute appendicitis.
- 10% of cases present with "Carcinoid syndrome" which is a syndrome of:

-Abdominal colic. -Bronchospasm. -Cardiac (tricuspid & pulmonary valve) dysfunction.

-Diarrhea. -Enlarged liver. -Flushing.

These manifestations are due to biologically active substances e.g. serotonin secreted directly into the systemic circulation by liver metastases or primary bronchial and gonadal carcinoid. Serotonin from GIT carcinoids has no effect because it is inactivated in the liver.

Investigations:

1- Imaging studies of the liver metastasis with needle biopsy of one of them.

3- Estimation of the daily urinary excretion of 5 Hydroxy Indole Acetic Acid (5 HIAA)

Normally < 10 mg /day (> 50 mg /day is diagnostic).

Treatment:

-For small tumours discovered at appendectomy:

-If it is at the tip, removal of the appendix is sufficient.

-If it is at the base, right hemicolectomy is indicated.

-For hepatic metastases, surgical excision or debulking is the best measure if possible.

Tumours of the colon

Benign tumours

A- Epithelial

1- Juvenile (retention) polyps:-

-They are pedunculated, usually single or few polyps covered with mucous.

-They are commoner in the rectum.

-They affect children and commonly cause bleeding per rectum but rarely, prolapse.

-They never turn malignant and may undergo autoamputation.

Villous adenoma

2- Villous adenoma:

-They are sessile with multiple frond like projections.

-Common sites are rectum & sigmoid colon.

-They affect patients over 45 years.

-They usually present with passage of blood & mucous per rectum.

-They may cause profuse watery mucoid diarrhea with loss of excessive amounts of electrolytes & protein leading to hypokalaemia & hypoproteinaemia.

-They commonly turn malignant.

3- Adenomatous polyps.

- They are the commonest tumours of the colon.
- They are usually pedunculated firm polypi.
- They are common in the rectum and sigmoid.
- They affect patients over 21 years.
- They are usually asymptomatic, but may cause bleeding, prolapse or intussusception.
- They often turn malignant.

4- Familial adenomatous polyposis (FAP):

- It is a hereditary disease (autosomal dominant trait).
- The polyps start to appear at puberty in the rectum and sigmoid colon. By the age of 21 year. The whole colon and rectum are studded with very large number of polypi.
- By the fifth decade, almost all patients develop carcinomatous changes.
- The symptoms appear after puberty with recurrent abdominal colic & diarrhea with blood and mucous, marked tenesmus, general weakness, anaemia and loss of weight.
- PR exam: may reveal rectal polypi.
- Barium enema: multiple smooth globular filling defects throughout the entire colon

giving honey comb appearance.

- Sigmoidoscopy and biopsy are mandatory.

5- Gardner's syndrome:

Familial adenomatous polyposis, multiple sebaceous cysts, multiple osteomata of the skull and mandible & desmoid tumours (especially of the abdominal incisions).

6- Turcot's syndrome:

FAP & CNS tumours (e.g. medulloblastoma or glioblastoma).

7- Hamartomatous polyps:

a- Peutz-Jegher's syndrome: a rare hereditary disease which consists of widespread intestinal hamartomatous polyposis accompanied by mucocutaneous pigmentations.

b- Diffuse juvenile polyposis (multiple GIT hamartomas and adenomas with 10% cancer risk).

B- Connective tissue tumours:-

Lipoma, haemangioma, leiomyoma and fibroma. They may give rise to bleeding per rectum or intussusception.

Treatment:

1- Juvenile and adenomatous polyps:-

They are best treated by proctoscopic or colonscopic polypectomy.The polyps are sent for histopathological examination (HPE.).

2-Villous polyps:

They are treated by complete local excision for histopathological examination. If malignant changes are detected, further radical resection is needed.

3- Familial adenomatous polyposis (and its variants) and diffuse juvenile polyposis:-

-They are treated by one of the following:

- Total coloproctectomy with terminal ileostomy or better ileal pouch-anal anastomosis.
- Total colectomy with ileoproctostomy before the age of 30 years, with regular endoscopic fulguration of rectal polypi.

**Sulindac* is NSAID and claimed to induce regression of rectal polypi after total colectomy.

Ileal pouch

-Recently, total colectomy with rectal mucosectomy and ileo-anal anastomosis through the rectal muscular cuff (*Soave like operation*) gives promising results.

Malignant tumours

Carcinoma of the colon

Aetiology

I. Premalignant conditions:

1- Adenomatous and villous polypi.

2- Familial adenomatous polyposis and Gardener's syndrome.

3- Ulcerative colitis and Crohn's disease.

II- Predisposing factors:

A- Genetic factors:

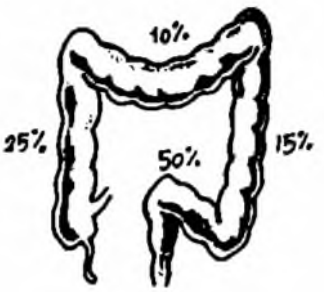
There is strong evidence of hereditary factors in pathogenesis of colon cancer.

B- Dietary factors:-

1-Increased fat intake: They provide the potent carcinogen polycyclic hydrocarbons .Also, they inactivate the benzpyrene hydroxylase enz. responsible for damage of the potent carcinogen benzpyrene.

2- Alcohol consumption.

3- Smoking



Site incidence

Pathology:-

Site: The commonest site is the sigmoid and rectosigmoid region (50%), caecum and ascending colon (25%).

MAC: There are three common types:

1- *The annular (stenosing) type*: it is common on the left side and forms a firm annular stricture encroaching on the lumen and infiltrating all coats.

2- *The cauliflower (polypoidal) type*: It is common on the right side and forms a fungating mass projecting into the lumen.

3- *The ulcerative type*: common in the caecum and has the malignant criteria (hard base, necrotic floor, irregular margin & raised everted edge).

Annular type

Cauliflower type

Ulcerative type

MIC: Adenocarcinoma with its different types.

1- *Columnar cell carcinoma.*

2- *Spheroidal cell carcinoma*: Scirrhous, encephaloid & anaplastic.

3- *Colloid adenocarcinoma*: Signet ring appearance (worst prognosis).

Spread:

1- Local spread: In the horizontal > the longitudinal direction (1/4 the circum. in 6 months).

2- *Lymphatic spread*: To the epicolic, paracolic, intermediate, then central group of mesenteric lymph nodes, which drain into the para-aortic LNs to cisterna chyli → thoracic duct. The left supraclavicular LN. (Virchow's gland) may be involved by retrograde spread.

3- *Blood spread*: (usually late) mainly to the liver. Lungs, bones & brain may also be involved.

4-*Transperitoneal spread* :(especially with mucoid carcinoma)

Malignant cells may spill over the serous surface to implant elsewhere in the peritoneal cavity (omentum, intestine, ovaries *(Krukenberg's tumour)* or rectovesical pouch (Blumer's shelf).

Complications :

1- Bleeding: is rare except in cancer of the right side.

2- Perforation: is rare and slow giving rise to abscess or fistula.

3-Acute intestinal obstruction is usually due to faecal impaction.

5- Anaemia, loss of weight, and general debility.

6- Distant metastases.

Incidence:

- It is common above 50 years but no age is immune.
- Cancer of the left side is commoner in males but ,that of the right side is commoner in females

Clinical picture:

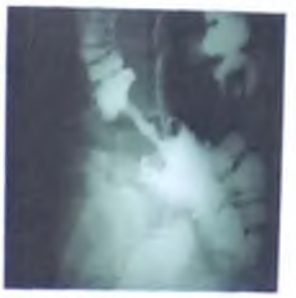
I- Carcinoma of the right colon:-

- Usually the patient complains of Anaemia, Anorexia and Asthenia (3 As).
- Abdominal colic in the right side of the abdomen may be followed by attack of diarrhea containing blood & mucous.
- Intestinal obstruction is rare (wide caliber - fluidy contents - nonstenosing type).
- Mass felt in the right side of the abdomen (the tumour).
- Picture of complications may be the first presentation.

II- Carcinoma of the left colon:-

- The patient complains of obstructive manifestations in the form of progressive constipation, usually interrupted by attacks of spurious diarrhea caused by accumulated mucous above the obstructing carcinoma.
- Acute on top of chronic intestinal obstruction is common due to faecal impaction.
- Palpable indentible mass in the Lt. iliac fossa is due to faecal impaction and not the tumour
- Picture of complications may be the first presentation.

Investigations:



Apple core appearance

1- Barium enema: often shows filling defect or constant irregular short segment stricture, with characteristic apple core appearance.

2- Double contrast enema: to delineate the wall of the colon and detect smaller lesions that couldn't alter the contour in ba. enema.

3-Colonoscopy: the most accurate method and enable to biopsy the tumour for HPE. Also, for detection of synchronous tumors

5- Estimation of S. Carcino-embryonic antigen (CEA): It is a glycoprotein "tumour marker ". It is elevated in most GIT cancers, some non-GIT cancers and some benign conditions. It is elevated only in about 70% of patients of colorectal cancer, so, it is neither diagnostic nor exclusive but rather prognostic for follow up of cases after surgery to detect early recurrence.

6- Abd. U/S, liver scan, CT scan, MRI: to detect metastasis.

Differential diagnosis:

-Carcinoma of the right colon should be differentiated from other masses in Rt. iliac fossa.

-From benign colonic lesions e.g. ulcerative colitis, diverticulosis, amoebiasis & Crohn's disease.

Treatment:

It depends upon whether the patient presented with acute intestinal obstruction or not.

I- Non-obstructive cases.

A- Operable: →Radical resection.

I- Pre-operative preparation:

1- Improve the general condition of the patient (tonics - vitamins- blood transfusion).

2- Colonic preparation (mechanical and chemical).

Aim: to clean, decompress and sterilize the colon.

a) NPO (only clear fluids may be allowed) + I.V. fluids.

b) Intestinal antiseptics, Neomycin - Metronidazole - Sulfa – Lactulose.

c) Repeated colonic lavage (enemata).

Rules in colonic surgery:

The safety margin for resection in the colon is 5 cm however the radical resection should include the draining LNs and this necessitates ligation-division of the major bl. vessels supplying the colon. This is the factor which determines the extent of resection rather than the safety margin i.e. we should resect the whole segment supplied by those vessels). In addition, resection should be planned so that anastomosis is done between two parts of the intestine with complete serosa e.g. ileum, transverse colon or pelvic colon. Fortunately, this factor correlates with the first factor.

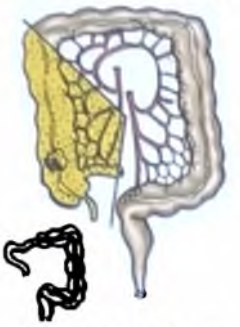
II- *Operative treatment:*

1- Carcinoma of the caecum & ascending colon: → Rt. hemicolectomy

We remove:-

- The last 10 inches of the ileum.
- Caecum & ascending colon.
- Hepatic flexure and proximal 1/3 of transverse colon.

Anastomosis is done between the ileum & transverse colon.



Rt. hemicolectomy

2- Carcinoma of the transverse colon: → Transverse colectomy.

We remove:-

- The transverse colon.
- Transverse mesocolon and greater omentum.

The anastomosis is done by end-to-end anastomosis.

Transverse colectomy

3- Carcinoma of the descending colon: → Left hemicolectomy.

Aim: To remove:-

- The descending colon.
- The splenic flexure and distal 1/3 of transverse colon.

Anastomosis is done between the transverse and sigmoid colons.

4- Carcinoma of the sigmoid colon: →Extended sigmoid colectomy.

We remove the descending & sigmoid colon.

The anastomosis is done between the transverse colon & the rectum.

Left hemicolectomy

5- Carcinoma of the hepatic flexure: → extended Rt. hemicolectomy

Rt. hemicolectomy and Transverse colectomy.

6- Carcinoma of the splenic flexure: → extended Lt. hemicolectomy.

Lt. hemicolectomy and transverse colectomy.

B. Inoperable cases.

Criteria of inoperability (see before). Solitary hepatic metastasis is not a sign of inoperability ,as it may be resected with 50 % cure rate.

Extended sigmoid colectomy

I - Palliative surgery: To avoid complications e.g. obstruction.

1- Resectable tumour: → palliative resection.

2-irresectable tumour: Rt. side → ileotransverse colostomy.

Lt. side → transverse pelvic anastomosis.

II- Radio therapy.

III- Chemotherapy (5 fluorouracil).

IV- Immunotherapy using monoclonal antibodies against tumour antigen.

II Obstructive Cases

I- Resuscitation: I.V. fluid. N/G suction ...etc.

II- Immediate laparotomy.

1- Resectable tumour :

- Fit patient: Right colon → right hemicolectomy.
  Left colon →1ry resection anastomosis after on table lavage (Dudley lavage).

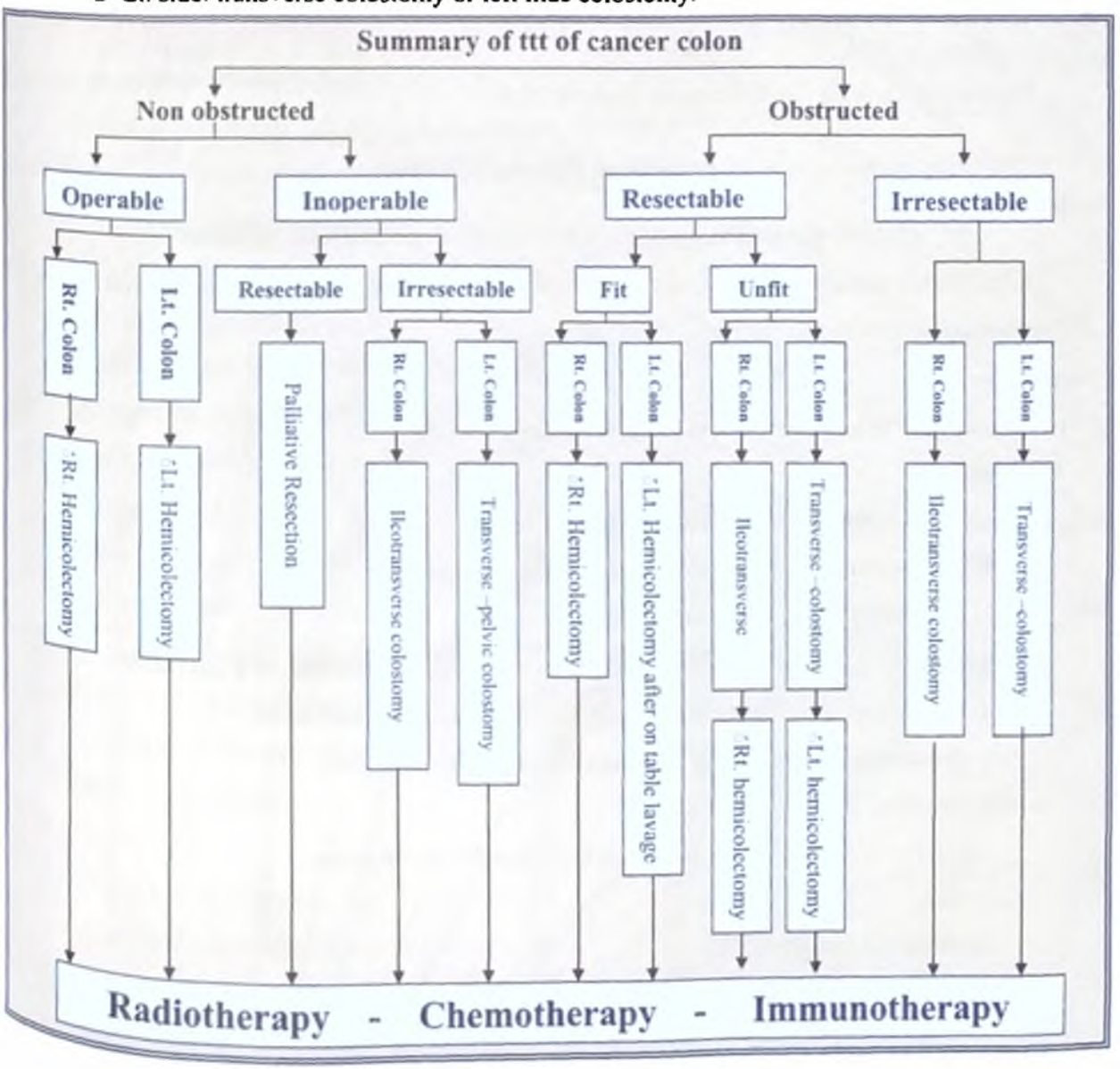
Transverse pelvic anastomosis

- Unfit patient: Right colon → ileo-transverse colostomy & later on. Rt. hemicolectomy.
  Left colon → transverse colostomy & later ,Left hemicolectomy.

2- Irresectable tumours.

1- Rt. side: ileotransverse colostomy.

2- Lt. side: transverse colostomy or left iliac colostomy.



Prognosis:

It depends on several factors:

1- *Stage of the disease*: according to Duke's classifications (see later).

2- *Hepatic metastasis*: the most important prognostic factor.

3- *Histopathological features*: mucoid carcinoma has the worst prognosis.

4- *Presence of complications*: e.g. obstruction & perforation.

5- *The site of the tumour*: has a little effect on prognosis because the Lt. side cancer is of high grade malignancy but presents earlier (obstructive) and spreads slower (poor lymphatics) .Also, more radical resection could be performed on the left side as the inf. mesentric artery can be ligated & divided. Rt. side cancer is of low grade malignancy but presents late, spreads earlier (rich in lymphatics) and less radical resection could be performed.

- The average 5-year survival rate for colorectal cancer is :

Stage A: 90% Stage B : 60% Stage C : 30% Stage D : 5%

The overall survival rate (all stages) is about 35 %.

Intestinal (Faecal) fistula

Definition. :

(Fistula means abnormal track between two epithelialized surfaces)

Faecal fistula means abnormal track between the intestinal lumen and skin delivering the intestinal contents to the exterior.

Aetiology:

Congenital: due to persistent patent vitello-intestinal duct.

Acquired:

A- Traumatic = Postoperative: The commonest cause.

1- Leakage at the suture line e.g. intestinal anastomosis.

2- Accidental injury to the intestinal wall.

3- Pressure necrosis by neglected drainage tubes, missed sponges, or instruments.

4- Operations on inflamed bowels e.g. Crohn's, T.B., and irradiation.

N.B. Colostomy and ileostomy are surgically made faecal fistula.

B-Inflammatory:

e.g. T.B., Crohn's disease, ulcerative colitis, and actinomycosis.

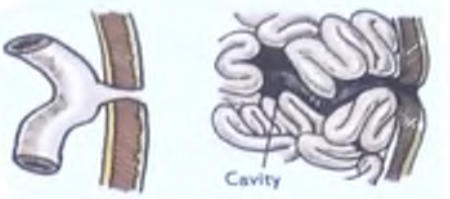
C-Malignant:

In advanced malignancy.

N.B: Fistula due to inflammatory malignant causes is usually called spontaneous fistula.

Pathology:

Types:



Direct fistula **Indirect fistula**

I- According to the length.

1-*Direct fistula*: with short track .It may be:

a-*Mucous lined* : which is lined by mucous membrane and never closes .

b- Granulation tissue lined: so it may close spontaneously.

2-*Indirect fistula*: with long tortuous track communicating an abscess cavity to a distant intestinal loop. It is usually partially epithelialized.

II- *According to the output:-*

1- *High output fistula*: Output is more than 500ml /24 h.

- It is usually connected to proximal bowel e.g. duodenum or jejunum.
- It results in much fluid and electrolytes deficit and malnutrition.
- In addition, the high digestive power of the discharge leads to skin excoriation.

2- Low output fistula: out put < 500ml /24h.

- It is usually connected to the distal bowel e.g. colon.
- Fluids & electrolytes deficit are minimal & nutrition is mildly affected.
- Skin excoriation is mild.

Complications:

1- Fluids and electrolytes deficit.

2- Malnutrition: hypoprotienaemia, anaemia, and avitaminosis with weight loss.

3- Skin excoriation and maceration.

Clinical picture:-

1-Fistula is usually preceded by manifestations of intra-abdominal abscess e.g. fever and abdominal pain.

2-The abscess then drains purulent or bloody discharge, and then intestinal contents begin to discharge from the fistula. High fistula usually discharges bile stained undigested food, while low fistula discharges semisolid faeces.

3- Stage of complications.

Investigations

I- To confirm the diagnosis: e.g. to exclude sinus with feculent pus.

1- *Charcoal test or methylene blue test*: taken orally discovered in discharge after few hours.

2- *Fistulogram* or barium follow through or enema. It also shows the length of the track, the loop of origin and distal obstruction if present.

II- To detect complications = to asses the general condition:-

Hb, bl. Electrolytes, S. albumin, CVP, liver and kidney functions.

III- To detect the cause:

1- Biopsy to exclude specific infections or malignancy.

2- Radiography: plain and contrast may show distal obstruction or specific pathology.

Treatment

I- Conservative treatment:

"Faecal fistulas tend to close spontaneously provided that the continuity of the gut isn't interrupted".

1- NPO or minimal residual diet.

2- I.V. fluids, bl. transfusion, parenteral nutrition may be needed in high fistulas and correction of electrolytes disturbance and avitaminosis.

3- Feeding jejunostomy: in duodenal fistula.

4- Protection of the skin by sump drain, & zinc oxide ointment.

5- Suppression of GIT secretions e.g. somatostadin (not properly evaluated).

II- Surgical treatment:

Indications:

1- Failure of conservative treatment.

2- Presence of distal obstruction.

3- Mucous lined fistula or presence of abscess cavities.

Operations:

Resection of the track together with the fistulous bowel segment and primary anastomosis.

Colostomy

Definition:

It is surgically made colonic fistula to form an artificial outlet of stool to the exterior.

Indications:

1. *Permanent*: after surgical resection of the rectum.
2. *Temporary*: in order to:

-Relieve distal obstruction e.g. cancer sigmoid, high imperforate anus Hirschsprung's disease.

-Deviate stool away from distal lesions or anastomosis e.g. perineal , rectal or colonic tears, perforated diverticulitis

-As part of exteriorization resection (Paul-Mickulicz operation)

Terminal colostomy

Types:

1- Terminal colostomy:

Where the distal end is brought out as abdominal anus.

2- Simple loop colostomy:

When a loop of the colon proximal to the lesion is brought out and opened to the exterior.

Simple loop colostomy

3- Double-barreled colostomy: = Paul Mickullcz op.

Where the two limbs of the proximal and distal ends are sutured to each other in order to form an intervening spur, which can be crushed later on by special crushing enterotome to communicate the two lumens as a step in its closure.

Double-barreled colostomy

4- Divine's (defunctioning) colostomy.

Where the proximal and distal ends brought separately to the surface with skin bridge in between to:-

- prevent contamination of the distal colon → sterilization.
- Give rest to the distal colon so, the vascularity decreases and resection will be easier if indicated.

Divine's colostomy

5- Caecostomy: (Rarely done nowadays).

For decompression of proximal colon obstruction.

A large caliber tube is inserted in the caecum and exteriorized through the RT. Iliac fossa to be removed later.

Caecostomy

Complications:

1- Prolapse of the colon like intussusception

2- Retraction

3- Ischaemia & necrosis

4- Stenosis

5- Hernia: It is either

- external when a loop of intestine herniates beside the colon through the abdominal wall
- internal when a loop of intestine herniates in the window between the colon and the lateral abdominal wall

Intestinal obstruction

Definition:

Failure of propulsion of intestinal contents due to either mechanical occlusion of the lumen (dynamic obstruction) or failure of the propulsive movement (adynamic obstruction).

Actiology:

I-Mechanical (Occluded lumen):

1-Simple obstruction: occluded lumen without interference with blood supply.

A-In the lumen:-

1-Meconium ileus
2-Gall stone ileus and bolus obstruction
3-Ascaris mass
4-Faecal impaction ,F.B., or enterolith

B-In. the wall:

-Congenital atresia

-Inflammatory stricture. e.g. T.B. Crohn's, ulcerative colitis, diverticulitis.

-Malignancy: cancer colon, and rectum.

C-outside the wall:

-Bands and adhesions (the commonest cause).

-Tumours and enlarged LNs.

2-Strangulated obstruction: There is interference with blood supply:

A-Strangulated hernia (external or internal).

B-Volvuolus.

C-Intussusception.

N.B: MVO is strangulated adynamic obstruction (i.e. patent lumen).

II Functional (adynamic) patent lumen:

1-Paralytic ileus.

2-Spastic ileus: Hirschsprung's disease is a good example.

3-Mesentric vascular occlusion (MVO) is an example of strangulated adynamic obstruction.

Pathology:

1-Proximal to obstruction:

The proximal loop passes through the following phases:

a--Hyperperistaltic phase.

B-Antiperistaltic phase.

C-Stage of dilatation & distension with fluids & gas.

* Fluids: swallowed, secreted & diffused from blood

The secreted part alone is about 8 liters
- 4000 ml above the pylorus
  - 1500 ml saliva
  - 2500 ml gastric juice
- 4000 ml below the pylorus
  - 1000ml bile & pancreatic sec.
  - 3000 ml intestinal secretion

* Gas
- swallowed (68%),.
- diffused from blood (22%).
- produced from putrefaction (10%) .

-Late: bacterial proliferation with breakdown of retained intestinal contents produces toxins that accumulate in the stagnant fluid and do not pass to the circulation except after release of obstruction, which may lead to toxaemia and may prove fatal.

2-Distal to obstruction:

Early: normal peristalsis to evacuate the residual content

Late: the distal segment is empty collapsed contracted and immobile.

3-At the site of obstruction:

I- Simple obstruction: The cause is obvious.

II- Strangulated obstruction:

-The strangulated loop becomes distended with gas & fluid.

-At first, the venous flow is occluded due to low pressure leading to oedema and congestion.

- Arterial flow is then occluded leading to ischaemia and gangrene.

-The devitalized wall of the intestine permits passage of toxins & bacteria to the peritoneal cavity & circulation causing toxaemia. Lastly, perforation leads to peritonitis.

Complications:

1-Hypovolaemic shock especially in strangulated obstruction due to additional blood loss into the strangulated loop.

2- Dehydration & electrolytes imbalance: hyponatraemia & hypokalaemia.

3-Toxaemia: in strangulated obstruction due to bacterial translocation...

4-Perforation & peritonitis.

Clinical picture:

Symptoms:

1-Pain: the earliest symptom:

A-Simple obstruction: colicky pain in attacks with longer free intervals.

B-Strangulated obstruction: colicky pain with short intervals with constant dull aching pain.

C-Paralytic ileus: No colic.

2-Vomiting:

The higher the obstruction, the earlier the vomiting.

3-Constipation:

-Absolute constipation to both faeces and flatus.

-The more lower the obstruction, the earlier the constipation.

4-Distension:

-The lower the obstruction the more is distension.

Signs:

I- General:

- Signs of dehydration, shock or toxaemia.
- General signs of the cause e.g. distant metastasis of GIT cancer.

II- Local = Abdominal exam:

A-Inspection:

-Abdominal distention.

-Visible peristalsis.

B-Palpation:

1-Simple obstruction: mild tenderness over the distended loops maximum over the obst site.

2-Strangulated obstruction: tenderness & rebound tenderness over the strangulated loop.

C-Percussion:

1-Hyper-resonance over the distended loops.

D-Auscultation:

-Loud exaggerated intestinal sounds in hyperperistaltic stage.

-Dead silent abdomen in paralytic ileus and stage of dilatation.

E- Per-rectal examination (PR):

- Empty rectum supports the diagnosis.

-It may reveal anorectal carcinoma or red currant jelly stool in intussusception.

*How to suspect the level of obstruction clinically?

Items	Proximal (jejunum)	Middle (ileum)	Distal (colonic)
1-Pain	above the umbilicus	Around & below it	Peripheral
2-Vomiting	-Very early (with pain)	-1-2h after pain	1-2 day after pain
	-Copious	-moderate	mild
3-Constipation	Late	Intermediate	Early
4-Abd. distension	-Mild or absent	-Inter mediate	-Marked
	-central	-central	-peripheral
5-Dehydration	Marked early	Intermediate	Mild & late

Investigations:

1-Double enema test:

Two enemas are given one hour apart. If the second enema comes without 3F (Faeces Flatus or Force), intestinal obstruction is proved.

2-Radiological investigations:

a- Plain X. ray abdomen erect position:

It reveals distended loops with multiple fluid levels in stepladder pattern.

- *Jejunum*: shows circular folds called "*valvulae connivents*".
- *Ileum*: shows shapeless characterless tubes.
- Colon: typical haustrations.

B-Barium enema:

When colonic obstruction is suspected.

3-Laboratory investigations:

A-Total leucocytic count: markedly rises in cases of strangulation.

B- Serum electrolytes: decreased sodium and potassium levels.

Differential Diagnosis:

1- From other causes of acute abdomen (see later).

2- Simple obstruction from strangulated one (see later).

Item	Simple obst.	Strangulated. Obst.	Paralytic ileus
1-Pain	Intermittent colicky pain with long free intervals.	Attacks of colicky pain with short intervals of constant dull aching pain.	mild or no pain
2-Shock	Mild.	Severe.	Moderate.
3-Palpation	Tenderness especially over the site of obstruction.	Tenderness and rebound tenderness.	Very mild tenderness.
4-Auscultation	Hyperperistalsis then silent abdomen.	Hyperperistalsis then silent abdomen.	Dead silent abdomen.
5-N/G suction	It relieves pain in hours.	Does not relieve pain.	Relieves distention.

Treatment:

1-Conservative treatment:

-*Indications*

1-Preoperative measures.

2-Some cases of adhesive intestinal obstruction.

-It includes:

1- Correction of fluid & electrolytes imbalance i.e. I.V. fluids according to the deficit.

We give Ringer lactate and glucose 5% & we add Kadalex is added only if the patient is hypokalaemic. Fluid chart is mandatory.

2- Nasogastric suction through Ryle's tube for:

A-Preoperative benefits

- It relieves distension, which may cause cardiac & respiratory embarrassment.
- It relieves congestion & oedema of the intestines and helps return of tone & peristalsis.

B- Operative benefits:

- For anaesthesia: it prevents vomiting & aspiration pneumonia.
- For surgeon: it provides easy manipulation & easy closure.

C- Postoperative benefits:

- It prevents massive toxic absorption after release of obstruction.
- It reduces the incidence of postoperative paralytic ileus.

3- Antibiotics: to guard against respiratory infection, peritonitis & septicemia.

II- Surgical ttt:

Apart from some cases of adhesive intestinal obstruction ,in which the previous conservative measures may be curative, most cases need emergency exploration.

Exploration: : midline is usually satisfactory.

1-Deliver the caecum and examine:

- If collapsed, it is small intestinal obstruction → follow the ileum to the distended loops.
- If distended, it is large intestinal obstruction → follow the colon to the collapsed part.

2-Decompress the bowels if greatly distended by:

- threading a long nasogastric tube through the intestine down to the site of obstruction.
-decompression through a small stab in the bowel and introduction of wide bore catheter connected to a sucker.

3-Deal with the cause:

-Simple obstruction: Remove the cause if possible or do bypass or colostomy.

-Strangulated obstruction: Remove the cause of strangulation and examine the viability of the loop.

Item		Viable loop	Gangrenous loop
-Inspection	-luster	Present (shining)	Absent (dull)
	-colour	Red (light)	Dark or black
	-peristalsis	Seen	Absent
-Palpation	-tone	Present (firm)	Absent (flabby)
	-Pulsation	Felt in the mesentery	Absent
Operative Doppler U/S		+ ve	-ve

If the viability of a loop is questionable, try to improve it by:

A) ↑↑ Oxygenation. b) Hot fomentation.

If not improved or proved gangrenous → resection of gangrenous loop.

A- Small intestine or right colon → 1ry resection anastomosis.

B- Lt. colon → either exteriorization resection ended by double barrel colostomy or recently primary resection anastomosis after on table colonic lavage (Dudely)

How to guess the cause of obstruction from the age of the patient?

1- Newborn (first month):

a -Congenital pyloric stenosis.

b- Malrotation or volvuolus neonatorum.

c- Congenital duodenal obstruction.

d- Jejuno ileal atresia or stenosis (the commonest cause).

e- Meconium ileus.

f- Duplication of the intestine.

g- Hirshsprung disease.

h- Imperforate anus.

2- Infancy (1 month - 2years) & Childhood (2y-12y):

a -Intussusception (the commonest cause).

b -Strangulated ext. hernia (the commonest cause) .

c-Ascaris mass obstruction .

3- Young adult and middle age:

A-Adhesive intestinal obstruction (the commonest cause).

B-Strangulated hernia.

C-Paralytic ileus.

D-Stricture obstruction e.g. T.B., or gall stone obstruction.

4- Old age:

a- Malignant obstruction (the commonest).

b-Volvuolus sigmoid.

c- Faecal impaction.

The commonest causes of intestinal obstruction as a whole are:

1-Adhesive intestinal obstruction.

2-Strangulated ext. hernia.

3-Malignant obstruction.

4-Paralytic ileus.

1- Ascaris mass obstruction

Aetiology:

Obstruction of the terminal ileum by aggregation of Ascaris lumbricoides worms usually following antihelminthic ttt.

Incidence:

Common in children below 10years in tropics.

Clinical picture:

- History of Ascaris infestation or intake of antihelminthics may be positive.
- The vomitus may contain worms.
- Picture of simple intestinal obstruction.

Investigations:

Leucocytic count will show marked oesinophilia.

Treatment:

-Conservative ttt: (N/G suction + I.V. fluids) may succeed.

-Exploration: The mass is identified and trial is done to knead it along the ileum to the colon without opening the bowel because the worms tend to eat their way through the catgut sutures to the peritoneum if the bowel was opened and sutured.

*If kneading fails, remove the mass through transverse incision sutured by silk suture.

2-Gall stone ileus

Aetiology:

Obstruction of the terminal ileum by large gall stone (2.5 cm in diameter or more) which had ulcerated through the gall bladder wall into the duodenum .It passes down to be impacted usually 2 feet from the ileocaecal valve causing simple intestinal obstruction.

Incidence:

Common in old obese multiparous females with long history of dyspepsia.

Clinical picture

- Clinical picture of simple intestinal obstruction :

*Usually there is a long history of ch. cholecystitis with recent exacerbation.

*The diagnosis is usually delayed because it resembles exacerbation attacks of gall bladder disease to which both the patient and her physician are accustomed to.

Investigations

1-Plain X ray of abdomen in erect position: It may show:

-The classic multiple fluid level.

-The stone may be sees.

-Gas in the gall bladder or biliary tree (diagnostic).

Gall stone ileus

Treatment: Surgical.

Exploration after good preoperative preparation:

-Try to crush the stone between fingers without opening the bowel.

-If failed, open the ileum above the stone, and remove it then close the incision transversely

-Avoid any manipulation in the region of the gall bladder, which may break down the cholecyto-enteric fistula & may causes external fistula.

3-Faecal impaction

Aetiology:

Obstruction of the distal colon and rectum by inspissated faeces .

Incidence:

Common in elderly bed ridden patients.

Clinical picture:

- Clinical picture of simple distal intestinal obstruction.

-Indentible abdominal mass may be felt in the Lt. iliac fossa or the faecal mass is felt P/R.

Treatment:

-Conservative measures + repeated enemata may succeed to loosen the mass and relieve obstruction.

-If failed ,anal dilatation under anaesthesia and manual removal of the mass is done.

6-Adhesive intestinal obstruction

Aetiology:

Peritoneal irritation →fibrinous exudate → fibrinous adhesions between adjacent intestinal loops.

They may resolve or change into mature fibrous tissue → fibrous adhesions.

The irritating causes may be (theories):

1- *Infection:* peritonitis, & T.B.

2- *Foreign bodies* e.g. talk powder, & silk sutures.

3-*Trauma:* mechanical or thermal e.g. diathermy or hot fomentations

4- *Vascular:* ischaemia or congestion.

Pathology:

Types: 1-Fibrinous adhesions: which is easily broken by blunt dissection.

2-Fibrous adhesions: which is firm and needs sharp dissection.

Clinical picture:

- There is a history of previous operation e.g. appendicitis or gynecological or past history of peritonitis or acute abdomen.
- C/p of simple obstruction but the adhesions may compress the blood supply and cause strangulated intestinal obstruction.

Noble's plication

Treatment:

I- Conservative ttt:

I.V. fluids, N/G suction may be beneficial and the intestinal movement may break down fibrinous adhesions.

Charle-Phillip's plication

II- Surgical ttt:

A-Exploration and division of the offending adhesions.

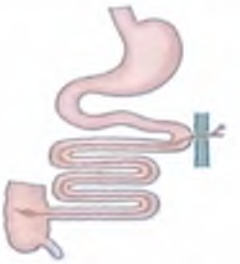
If adhesions are extensive → bypass by lateral anastomosis.

B-Prevention of recurrence:

1-Instillation of different substances e.g. hyaluronidase, heparin, steroids, fibrinolysin, dextran…etc. is usually useless.

2-*Noble's plication*: The adjacent loops are sutured along their antimesentric border in ordered fashion.

3-Charle-Phillip's transmesentric plication: Passing non-absorbable sutures in the mesentery few centimeters from the bowel that looks like a pouch of sausage



Baker's tube

4-Baker's tube: intraluminal tube splinting the loops in gentle curves removed 12 days later.

Intussusception (إنغلاف معوي)

Definition:

Invagination of a bowel segment into an adjoining one, which is usually distal.

Aetiology:

Partial obstruction is the main predisposing factor as it stimulates hyperperistaltic movement that forces the wall of a segment (intussusceptum) to invaginate into another (intussuscepient).

1-Infantile intussusception:

A-Idiopathic type (95%): *No organic cause.*

1-Predisposing factors:

A-Bulky ileocaecal valve.

B-Wide ileocaecal angle.

C-Mobile caecum and ascending colon.

D-Bloodless fold of Traeves : between the mesentry of ileum & caecum .

2-Precipitating factors:

Swelling of Payer's patches due to inflammation or hyperplasia causes partial obstruction and initiates intussusception. Swelling in Payer's patches may be due to:

1-Gastroenteritis at the time of teething & weaning due to change in intestinal flora.

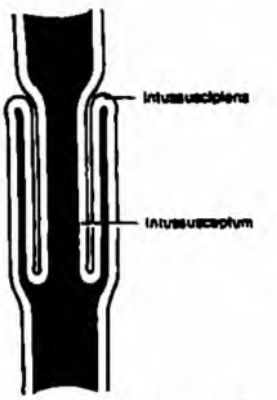
2-Respiratory infection by adenovirus → swelling of Payer's patches.

B-Organic type (5%):

Organic cause is detected as polyp or Meckel's diverticulum.

II- Adult intussusception:

-Usually there is an organic cause as tumours or parasite.



Composition of intussusception

Pathology:

**Composition*: It is composed of three layers.

-inner layer → entering layer.

-middle layer → returning layer.

-outer layer → unsheathing layer→ intussuscepient.

Types:

1-Ileocaecal: where the ileo caecal valve forms the apex of intussusception.

2-Ileocolic: where the ileocaecal valve forms the neck of intussusception.

3-Ileo-ileal.

4-Ileo -ileo colic.

5-Colo-colic.

6-Retrograde (jejunogastric) intussusception.

*Multiple intussusceptions occur in 1% of cases.

Ileocaecal **Ileocolic** **Ileo-ileal** **Ileo-ileo colic** **Colo-colic** **Jejunogastric**

***Pathophysiology:**

-In addition to luminal obstruction, the bl. supply of the intussusceptum is also impaired due to compression of its mesentry → strangulated intestinal obstruction.

-The ileo colic variety is the most vulnerable type to early gangrene as the ileo caecal valve forms a tight neck over the intussusceptum.

Complications:

1-Intestinal obstruction with all its complications.

2-Gangrene, perforation, & peritonitis.

Incidence:

-It has a seasonal incidence related to upper respiratory tract or gastrointestinal tract infection.

-It is more common in ♂ between 6-9 months (weaning) .

-The patient is usually well nourished.

Clinical picture:

Symptoms: (told by the mother):

1- Pain: - sudden onset of abdominal pain, the infant draws up his legs and screams. It lasts few minutes and recurs about every 15 minutes.

2- Vomiting may occur early with the onset of pain or late due to obstruction.

3- The patient often presents late with complication. Rarely, the intussusception may protrude through the anus.

Signs:

1-Mass: a sausage shaped mass is often palpable below the costal margin with its concavity towards the umbilicus due to traction on the ends of intussusception by the mesentry.

2 - *Dance's sign*: sense of emptiness of the right iliac fossa (ascended caecum).

3-PR exam: may reveal red currant jelly stool or the apex of intussusception.

Investigations:

1-Barium enema: shows barium arrest with characteristic *claw sign*.

Differential Diagnosis:

1-Gastroenteritis & enterocolitis: diarrhea with faecal matter and bile are always present.

2-*Henoch Schonlein's purpura*: a syndrome of purpuric rash, abdominal pain, bleeding per rectum, arthritis, and nephritis.

3-Rectal prolapse, probe test is diagnostic (see before).

Claw sign

Treatment:

I- Preoperative measures:

-I.V. fluid -N/G suction -Antibiotics

II- Reduction by hydrostatic pressure:

Using saline under U/S guide or barium enema under radiological guide under anaesthesia in the operating theatre (risky).

III- Operative reduction:

-Exploration through transverse supraumbilical incision.

-Try to reduce the intussusception in situ by milking the apex proximally. Do not pull on the proximal segment as it is ischaemic and may rupture

-If this failed, try again after pressure with hot fomentation for a sometime to reduce oedema.

-If failed, try *Cope's method*. Insert the little finger into the neck to dilate it, break adhesions, and try again.

Reduce the intussusception by milking the apex proximally

-If failed, open the ensheathing layer and reduce the intussusception. If viable, close the ensheathing layer. If gangrenous, do Rt. hemicolectomy and ileotransverse colostomy.

Volvuolus (إنفتال)

Definition:

It is axial rotation of a portion of the alimentary tract around two fixed points.

Types: 1-Volvulus of the stomach.

2-Volvulus of the small intestines.

3-Volvulus of the midgut (Volvuolus neonatorum).

4-Volvulus of the caecum.

5-Volvulus of the sigmoid colon.

Volvuolus

1-Volvulus of the small intestine

Aetiology

Commonly due to adhesions or bands between the antimesentric border of the loop and the parities (Meckel's diverticulum), or female pelvic organs.

Site: It occurs more commonly in the lower ileum.

Pathology, clinical picture & investigations

They are those of strangulated intestinal obstruction

Treatment:

1-Preoperative: N/G suction. 2- IV. Fluid. 3- Antibiotics.

2-Exploration: untwist & examine intestine. If not viable → primary resection anastomosis.

2-Volvulus of the caecum

Aetiology:

Abnormally hypermobile caecum and ascending colon.

Pathology: -

-It is more common in young adult female.

-The Volvuolus occurs usually in *the clockwise direction* obstructing both the ascending colon and the terminal ileum. The caecum usually moves out of the right iliac fossa.

Clinical picture

That of strangulated intestinl obst. + a tympanitic mass may be palpated in the mid abdomen.

Treatment:-

Exploration: The ballooned caecum should be decompressed first by insertion of a needle then try untwisting (usually in the anticlockwise direction) then examine the caecum:

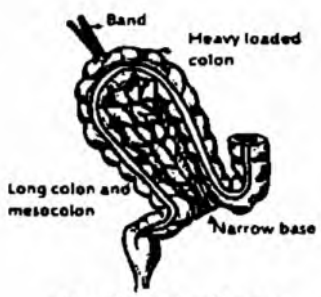
-If viable, do caecostomy to decompress the bowel and fix the caecum.

-If non-viable, Rt. hemicolectomy and ileotransverse anastomosis is advisable.

3-Volvulus sigmoid colon

Definition:

Axial rotation of the sigmoid colon around its mesenteric axis producing strangulated intestinal obstruction.



Predisposing factors

Aetiology:

1-Predisposing factors:

A-Dolichocolon: long pelvic colon with tall mesocolon.

B-Narrow attachment of the pelvic mesocolon.

C-Over loaded pelvic colon with faeces due to habitual constipation.

D-Presence of bands between the antimesentric border & parities.

2-Precipitating factors:-

1-Straining: the Volvuolus usually occurs while the patient is straining at stool.

2-Pregnancy: may be due to elevation of the colon by the pregnant uterus.

Pathology: -

Anticlockwise rotation

-The Volvuolus usually occurs in anticlockwise direction resulting in obstruction of the pelvic colon at its both ends "*closed loop obstruction*". The loop becomes hugely distended with gases & fluids.

-Rotation also compresses the bl. vessels. When it turns 1½ turn, the veins are occluded and the loop becomes congested.

-When the loop turns more, the arteries become also occluded causing ischaemia → gangrene → perforation → peritonitis.

Volvuolus

Complications:

1- Acute intestinal obstruction with all its complications.

2- Gangrene, perforation, & peritonitis.

3- Complications of surgery in elderly patients.

Incidence:

Common is elderly males specially those with habitual constipation e.g. bedridden.

Clinical picture:

I-Symptoms:

-Pain: sudden severe abdominal pain usually during straining.

-Distension: is early & marked.

-Hiccough: due to irritation of the under surface of the diaphragm.

-Constipation: is very early.

Ace of spades appearance

III-Signs:

-*Inspection*: marked abdominal distension.

-*Palpation*: a large cystic tender mass with rebound tenderness.

-*Percussion*: tympanitic hyper-resonance over the mass.

-*PR*: empty edematous rectum and blood on the gloves due to kink of the sup. rectal vein causing congestion of the rectum.

-Signs of complications.

Investigations:

1-Plain X-ray abdomen: single greatly distended loop of the bowel and smooth wall (loss of haustrations) rises up out of the pelvis.

2-Barium enema: shows barium arrest at the recto sigmoid junction with characteristic *"ace of spades "*or" *bird's peak deformity"*.

3-Leucocytic count: markedly elevated.

Differential diagnosis:

From other causes of large bowel obstruction e.g. cancer rectum.

Treatment

I- Preliminary resuscitation:

I.V. Fluids. N/G suction. Antibiotics.

II- Endoscopic decompression:

In early cases and with endoscopic aid & the patient is put in the knee elbow position and a sigmoidoscope is introduced into the twisted colon to decompress it. If the try was successful, elective resection can be done later.

III- Surgery:

Indications:

1-Failure of proctoscopic decompression.

2-Complications e.g. gangrene perforation.

3-Elective resection after successful decompression.

Procedure:-Exploration

- Try to untwist the bowel (usually in the clockwise direction). Meanwhile, an assistant advances a rectal tube through the anal route, guided by the surgeon to deflate the colon
- If the pelvic colon is viable, it is repositioned in the abdomen
- If the pelvic colon is non-viable, resection is done.

* Resection is done by either:

1-Paul Mickulicz operation: exteriorization resection ended by double barrel colostomy.

If the distal limb is too short to be exteriorized, it is closed as in Hartmann's operation for later anastomosis.

2-Resection with Iry anastomosis after:

A- On table colonic lavage.

B- Intraluminal colons bypass tube (coloshield).

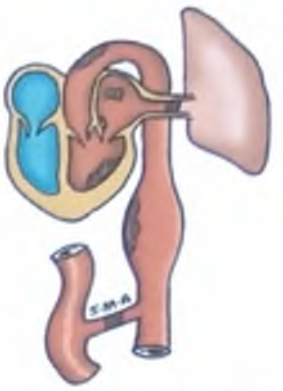
Paul Mickulicz operation

- Many surgeons advise resection of the colon even if it is viable as the recurrence rate is high (>50 %) and different prophylactic maneuvers against recurrence as colopexy or plication of the mesocolon are usually useless.

Mesenteric Vascular occlusion

Definition:

Strangulated adynamic obstruction due to occlusion of mesenteric artery or vein with patent intestinal lumen .



The source of emboli

Aetiology:

I-Mesenteric artery occlusion:

A-Embolism (commonest):

The source of the embolus (see before).

B-Thrombosis:

Usually on top of atherosclerosis.

II -Mesenteric vein thrombosis

Due to portal hypertension, portal pyaemia, sickle cell anemia and hypercoagulable states.

Pathology: -

-Whether the occlusion is arterial or venous, red infarction always results.

-The bowel: It is cyanosed and swollen with no peristalsis. The serosa is lusterless and the mucosa sloughs and bleeds. Finally, gangrene occurs.

-The peritoneum: blood stained fluid pours also in the peritoneum and the gangrenous bowel wall permits the passage of luminal organisms to the peritoneal cavity → septic peritonitis

Complications:

1-Shock is severe and rapidly develops due to loss of blood into both lumen and peritoneal cavity. Later, septic shock ensues due to gangrene & peritonitis.

2- Bleeding per rectum.

3- Gangrene, perforation & peritonitis.

Incidence:

Common in middle age or elderly patients usually with cardiovascular problem.

Clinical picture:

-History:

Most patients give history of a cardiovascular problem.

-Symptoms:

-Pain: sudden severe abdominal pain.

-Vomiting: repeated and may be with haematemesis.

-Bleeding per rectum: may be only after enema.

-Shock: rapidly develops.

-Signs:

-Generally: signs of shock and pallor.

-Abdominally: -Tenderness, early mild, late marked with rigidity & rebound tenderness.

-Silent abdomen on auscultation.

-PR: may be blood on gloves.

-Signs of complications may appear.

Investigations:

1-Plain X-ray abdomen: nonspecific finding.

2-Leucocytic count: markedly elevated > 15.000.

Differential Diagnosis:

From other causes of acute abdomen especially internal hemorrhage.

Treatment

1-Preoperative measures: -Resuscitation with I.V. fluid & bl. transfusion.

- N/G suction - Antibiotics.

2-Operative: exploration.

I- *Small branch occlusion* (short segment affection): -

1ry resection anastomosis (up to 70% of the small intestines can be resected in adults without serious metabolic troubles.

II- *Main stem (of SMA) occlusion* (long segment affection):

1- Viable intestines: → Attempt for reperfusion by direct arterial surgery e.g. mesenteric embolectomy, thrombendarterectomy. Second look operation is done12-24 hours later to ensure arterial patency & intestinal viability.

2- Non viable gut → resection may extend up to transverse colon with high mortality.

Paralytic ileus

Definition:

Loss of intestinal peristalsis leading to functional (adynamic) obstruction.

Aetiology:

Inhibition of the Auerbach's (Myenteric) plexus caused by:

1-*Reflex inhibition:* due to sympathetic stimulation after operations, trauma, or labour.

2-*Toxic inhibition:* as in peritonitis, typhoid fever & uraemia.

3-*Hypoxic inhibition*: hypoxia under anaesthesia.

4-*Biochemical inhibition*: electrolytes disturbance e.g. hypokalaemia & hyponatraemia.

5-*Mechanical inhibition*: by early fibrinous adhesions.

Pathology: -

Inhibition of the nerve plexuses →loss of intestinal peristalsis & muscle tone → distension with fluid & gas. The distension exerts more inhibition on intestinal motility → vicious circle.

Complications :-(causes of death):

1-Dehydration and shock.

2-Electrolytes disturbance e.g. hyponatraemia & hypokalaemia.

3-Burst abdomen.

Clinical picture:

Symptoms

-Most cases occur after operations especially those done for diffuse or pelvic peritonitis.

-Paralytic ileus is diagnosed when the following occurs 24 hours postoperatively:

A- No abdominal colic. B- Absolute constipation.

C-Abdominal distension. D- Copious vomiting.

Signs:

-Generally: Dehydration ± shock and signs of other complication.

-Abdominal: *There is marked distension with no visible peristalsis.

*Tympanitic hyper resonance.

*Dead silent abdomen.

Investigations:

1-Double enema test: the first enema may be retained or passes without feces or force.

2-Plain X ray abdomen erect: multiple fluid level.

Treatment:

1-Causal ttt: Removal or treatment of the aetiological factor.

2-Wide nasogastric suction: to break the viscous circle.

3-Resuscitation: by I.V. fluids & correction of electrolytes disturbance.

4-Drugs:-

A-Peristalsis stimulant e.g. prostigmin are contraindicated (Do not flog the tired horse).

B-Morphia may be given to restore muscle tone.

C-Prokinetic drugs e.g. Cisaprid have a promising effect.

5- Operation:

It is only considered if paralytic ileus persists for more than 5days. and mechanical obstruction is suspected. If exploration is negative. a long nasogastric tube is manipulated as down as possible to ensure good decompression.

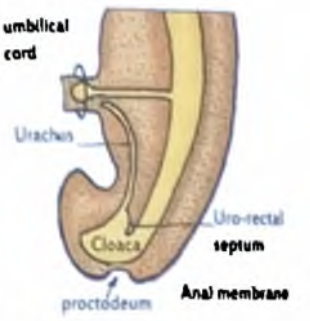
The Rectum & Anal Canal

Embryology

The uro-rectal septum divides the colaca into *uro-genital sinus* ventrally & *anorectal sinus* dorsally.

The rectum develops from the upper part of the anorectal sinus.

The upper 2/3 of the anal canal develops from the lower part of the anorectal sinus, while the lower 1/3 develops from *the proctodeum which is* an ectodermal upgrowth that meets the anorectal sinus at the anal (proctodeal) membrane that later ruptures making anal canal open to the exterior.



Development of the rectum and the anal canal

Anatomy of the rectum

-It is the direct continuation of the colon.

-It is about 15cm long (6 inches).

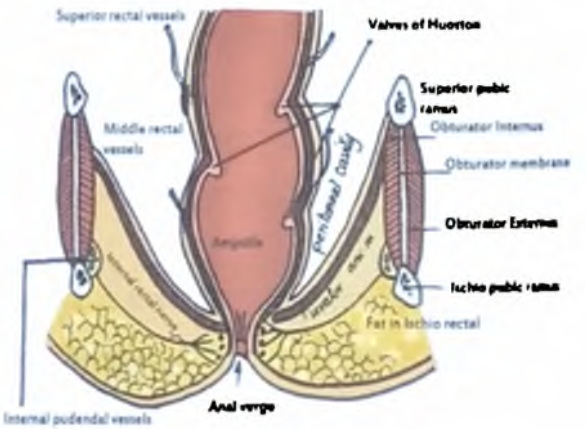
Begins: opposite third sacral piece (S_3).

Ends: at the level of the pelvic diaphragm, 1cm in front and below the tip of the coccyx.

Curvatures of the rectum:

1-One anteroposterior curve following the concavity of the sacrum.

2- Lateral 3 curves: The 1st to the right, the 2nd to the left then the 3rd to the right. These lateral curves are marked from within the rectum by three folds of the mucous membrane forming *valves of Houston*. Two small on the left and the larger and most constant one is on the right (the middle one).



Anatomy of the rectum

Fascial and ligamentous supports of the rectum:

1- *Fascia of Waldeyer*: from lower sacrum & coccyx to the lower part of the posterior rectal wall

2- *Lateral ligament of the rectum*: around the middle rectal vessels from the posteriolateral wall of the pelvis to lat. aspects of the rectum.

3- *Denonvilliers fascia*: rectovesical fascia between the prostate and seminal vesicles anteriorly and the ant. wall of the rectum posteriorly.

Blood supply.

Arterial:

1- Superior Rectal artery: is the continuation of the inferior mesenteric artery.

2- Middle rectal artery from the internal iliac artery.

3- Inferior rectal artery from the internal pudendal artery from the internal iliac artery.

Venous:

1- Superior rectal vein: continues as the inferior mesentric vein (portal).

2- Middle rectal veins end in the internal iliac vein. } (systemic)

3- Inferior rectal veins end in the internal iliac vein. }

Lymphatic drainage:

1- From the upper 2/3 of the rectum: lymphatics ascend along the sup. rectal vessels through the pararectal LNs to reach the inf. mesenteric and para aortic LNs.

2- From the lower 1/3 of the rectum lymphatics pass laterally along the middle rectal vessels through the pararectal LNs to the internal iliac LNs.

Nerve supply:

1- Parasympathetic: by the pelvic splanchnic nerve (S 2, 3, 4)

2- Sympathetic: around the inf. mesenteric and sup. rectal arteries.

Anatomy of the anal canal

-It is the direct continuation of the rectum.

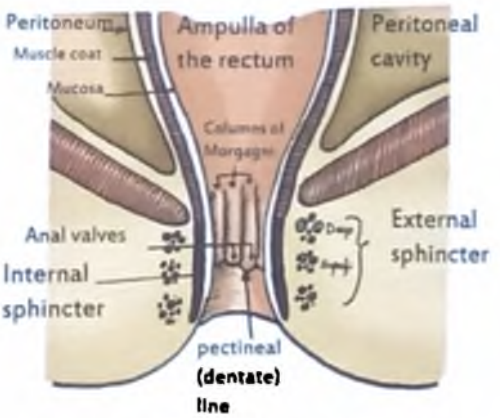
-It is about 4 cm in length.

Begins: 1 cm in front and slightly below the tip of the coccyx (anorectal junction).

Ends: in the anal verge (to the exterior) = anus.

Internal appearance of the anal canal:

- The upper 2/3 of the anal canal is lined by mucous membrane (endodermal in origin) while the lower 1/3 is covered by anoderm (modified skin) as it is ectodermal in origin.



Anatomy of the anal canal

- The mucous membrane of the upper 2/3 is thrown into 8-12 longitudinal folds called *columns of Morgagni* .Each column contains a terminal radicle of sup rectal art and vein.
- Lying at the lower end of columns of Morgagni are *the anal valves of Ball* which are small crescentic folds of mucous membrane. Above each valve, a small recess called anal sinus

or *Crypts of Morgagni* exist and into which the anal glands opens.

- The line where the anal valves lie is called the pectineal line or the dentate line, which marks the junction between the anoderm and ectoderm.

- The sphincters of the anal canal:

1- Internal sphincter: It is the direct continuation of the circular muscle layer of the rectum. It is involuntary muscle supplied by autonomic nerve fibers and its tone is the most important factor in keeping the canal closed (continent).

2- External sphincter: it is formed of three parts (rings), subcutaneous, superficial & deep part from down upwards. It is voluntary muscle supplied by somatic nerves (inf. rectal and perineal br. of the 4th sacral nerve).

Blood supply:

Arterial: branches from the sup., middle and inf.rectal arteries (the most imp is the sup. rectal).

Venous: into the sup. rectal vein (portal), middle and inf. rectal veins (systemic) .

Nerve supply:

1-Upper 2/3 (above the dental line) is supplied by autonomic system like the rectum.

2-lower 1/3 (below the dental line) by somatic fibers from the inf. rectal n. & perineal branch of S4.

Lymphatic drainage:

1- *Upper 2/3:* drains with the rectum through the pararectal LNs. to reach the int. iliac and inf. mesentric LNs. then to the para-aortic LNs.

2- Lower 1/3: downwards direction to superficial then deep inguinal lymph nodes.

Applied anatomy

1- Anorectal ring and angle: the junction between the rectum and anal canal forms an obtuse angle (120°) which straightens during defecation. Anorectal ring is formed by the joining of

- Puborectalis muscle
- Deep ext. sphincter
- Conjoined longitudinal m.
- Highest fibers of int. sphincter.

Division of the anorectal ring results in permanent incontinence.

2 -Denonviller's and Waldeyer's fascias are important factors in maintaining the rectum in position. Also they act as a barrier for malignant spread.

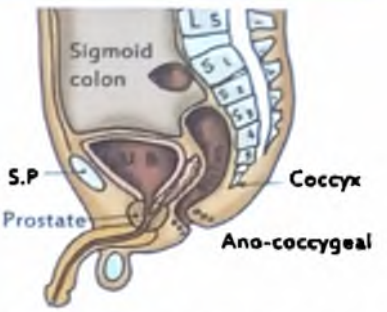
3 -The anal canal and lower rectum constitute one of the portosystemic anastomoses between sup. rectal vein (portal) and middle inf. rectal veins (systemic). In portal hypertension, dilatation and varicosites of these anastomotic veins may form, causing piles.

4- *The anterior relation of the rectum differs in the two sexes:*

Male	Female
1- Rectovesical pouch	1- Rectovaginal pouch
2- Post wall of the bladder & seminal vesicle	2- Uterus
3- Post surface of the prostate	3- Post wall of the vagina
4- The two vas deference & ureters	

This is important in spread of carcinoma.

5-The most important lymphatic pathway for malignant spread of the rectal carcinoma is the upward pathway along the sup. rectal and inf. mesentric vessels which should be removed on radical resection of these tumours. However, if the usual upward pathway is blocked by malignant infiltration, the flow may reverse to the lateral and downward directions (pelvic & inguinal LNs).



Sagittal section of the male pelvis

6-The importance of Houston's valves is that they may mask or hide small lesions during endoscopic exam.

7-The pectineal or dentate line marks the junction between the anorectal (endoderm) and proctodeum (ectoderm) so, there is marked difference above and below it :

Sagittal section of the female pelvis

Item	Above the dental line	Below the dental line
Origin	Hind gut (endodermal)	Proctodeum (ectoderm)
Lining	Mucosa	Modified skin
Epith	Cubical epith	Stratified squamous epith
Venous drainage	Portal	Systemic
Lymphatic	Upwards (sup. rectal)	Downward (inf. rectal)
Nerve supply	Autonomic (pelvic nerve)	Somatic (inf. rectal & perineal br. of S4)

8- Anal glands: are 8-12 racemose apocrine glands present in the intersphincteric plane and their ducts open in the anal sinuses or crypts above the anal valves. They are supposed to be the origin and route of spread of anorectal infection and suppuration.

I- Congenital diseases

Anorectal anomalies = Imperforate anus

Aetiology:

Failure of fusion of the recto anal gut (endoderm) with the proctodeum with or without failure of complete division of the cloaca into urogenital sinus and anorectal sinus by the urorectal septum

Pathology

Types:

According to whether the rectum passes through the pelvic diaphragm (levator ani) or ends above it, there are two types.

1- Low anomalies (infralevator or translevator type):

a- Covered anus: the anal canal is covered by a bar of skin with a track filled with meconium running forward to the perineal raphe.

b- Ectopic anus: the anal canal may open anteriorly in the vistibule or rarely in the vagina in ♀ and less commonly in the perineum in ♂.

c- Stenosed anus: the anus is normal in position but minute.

d- Membranous anus: normal in site, but, covered by thin membrane.

In low anomalies, the anal sphincters and pelvic floor are well-developed and so, anal continence after surgery is usually good.

2- High anomalies (supralevator types):

A-Rectal atresia: the anal canal is normal but ends blindly just below the pelvic floor and the rectum ends blindly above it.

B-Anorectal agenesis: the rectal pouch ends above the pelvic floor and usually there is a fistulous track into the bladder neck in males or less commonly into the vagina in females.

C- Cloaca: It occurs only in females where the bowel, urinary & genital tracts all open into a common wide cavity with one perineal opening.

Complications:

1- Intestinal obstruction.

2- Complication of fistula: e.g.urinary tract infection.

3- Complication after surgery: incontinence, stenosis, and impotence.

4- Associated congenital anomalies are common (remember VACTERL)

Covered anus

Ectopic anus

Stenosed anus

Membranous anus

Rectal atresia

Anorectal agenesis

Cloaca anomaly

Incidence

♂: ♀ = 2: 1

Clinical picture:

- Presents at birth by absent anus and failure to pass meconium.
- Minute opening (stenosed anus) or only small dimple at the site of absent anus may be noticed.
- Ectopic anus may be discovered in perineum or vulva or meconium may be noticed to drip continuously from the urethra (rectourethral fistula).
- Complications :distension, respiratory failure ,rupture caecum etc...

Invertogram in high anomaly

Investigations:

1- Radiography: (Invertogram):

- It is only done if there is no anus and no meconium (from vulva, urethra, or ectopic anus) for 24 h.
- The baby is held upside down with the site of the absent anus is marked by a coin or better metal marker then radiographed in the lateral position.

  If the distance between the gas in the distal bowel and the marker (the future anus) is more than 1 inch it is high anomaly and if it is less than 1 inch it is low anomaly OR If the distal bowel gas is proximal to pubococcygeal (PC) line, it is high anomaly. If it is distal to (PC) line, it is low anomaly.

2- Electromyography: to detect presence of ext. anal sphincter.

3- Urine culture: if fistula is suspected presence of proteus or pyocyaneous indicate recto-urinary fistula.

Treatment:

I- Low anomalies: *Usually simple problem treated by minor procedure in newborn*

1. *Covered anus*: open the track with scissors then repeated dilatation.
2. *Ectopic anus*: plastic cutback followed by perineal proctoplasty at the age of 3 months.
3. *Membranous anus* : cruciate incision.
4. *Stenosed anus* : repeated dilatation.

II- High anomalies:

Usually a major problem which needs major procedure so, it may be treated in two stages:

Stage I: colostomy. (Mandatory in rectourinary fistula.

Stage II: Rectal pull-through or Posterior Sagittal Anorectoplasty (PSARP) is performed at the age of 6 M. or when baby weight 9 kg.

How to differentiate between low anomalies and high anomalies?

1- Presence of anal dimple denotes low anomaly, while flat buttocks means high anomaly.

2- Presence of well-developed sphincter suggests low anomalies i.e. presence of corrugations at anal site increased with pinching.

3-Meconium emerging from tiny opening in perineum or base of penis means low anomaly while meconium from urethra or vulva means high anomaly.

4- Invertogram & electromyogram See before

Why differentiation is important?

- Crutiate incision in high anomalies will violate continence and complicate fistula repair.
- Colostomy in low anomalies is malpractice.

What are the fallacies of the invertogram?

1- Baby is less than 24 hours → false high. (Gas did not reach distal rectal pouch).

2- Viscid meconium (meconium plug → false high).

3- Crying or straining during radiography → false low.

4-Baby is not positioned correctly. X beam is not be centralized over the greater trochanter.

5-The baby is not kept in upright position for proper time (at least 5 min).(risky)

Anal fissure

Definition:

It is a longitudinal ulcer of the anoderm.

The fissure triad

Aetiology:

I- Indirect trauma = overstretch of the anoderm.

1- Overstretch of the anal canal by passage of hard bulky stool that tears the anal canal usually posteriorly (commonest cause).

2- Overstretch of the anoderm during labour usually anteriorly.

3- Pulling on the anoderm by large prolapsed piles.

II- Direct trauma:

1- F.B Trauma.

2- Injury by enema nozzle or during endoscopy.

3- Trauma of operation e.g. haemorroidectomy.

III- Specific pathology.

1- Crohn's disease 2- Ulcerative colitis 3-Venereal diseases

Pathology:

Site: It is most commonly in:

1- The midline posteriorly (90% overall), Due to:

❶- Pressure trauma lies on the post. wall due to the sharp backward turn of the anal canal.

❷- The anoderm is less supported by sphincter posteriorly.

❸- Poor vascularity and less elasticity.

2- The midline anteriorly (10% of ♀ patients) due to stretch of the anterior wall during labour.

3-Lateral anal fissure raises the suspicion of specific pathology.

Types:

1-Acute anal fissure: accompanied by inflammatory oedema and sphincteric spasm.

2-Chronic anal fissure: acute fissure tends to persist and become chronic due to:

1- Persistent spasm of the sphincter which impairs vascularity & drainage.

2- Continuous trauma & contamination by stool.

3-Pain causes constipation → hard stool → fissure→ pain (vicious circle).

The fissure triad ❶-*The fissure* becomes wider, deeper with fibrosed base.

❷-*Sentinel pile*: hypertrophied skin tags at the lower end of the fissure.

❸-*Anal papilla:* hypertrophied anal papilla at the upper end of the fissure.

Complications: 3 Ps.

1- Persistence = chronicity.

2- Perianal infection (perianal abscess and fistula).

3- Pruritis ani and eczema of the skin.

Clinical picture:

Symptoms:

I- Local symptoms:

1-Pain: Sharp severe pain with defecation which may drive the patient to constipate.

2- Bleeding: usually slight.

3- Pruritis: due to irritation by discharge.

II- Reflex symptoms: Dysuria, Dyspareunia or Dysmenorrhea.

Signs:

I- Acute fissure: (seen not felt).

1- Soft, Severely tender, superficial crack

2- Exaggerated anal corrugation due to sphincteric spasm.

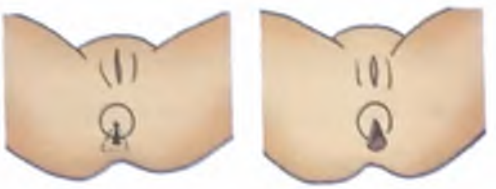
3- PR. exam is extremely painful and even impossible.

II- Chronic fissure examination may reveal the fissure triad.

1- Sentinel pile.

2- Longitudinal, indurated, tender ulcer.

3- Hypertrophied anal papilla.



Dorsal fissurectomy and sphincterotomy

Treatment:

I- Conservative ttt: for acute fissures.

1- *Avoid constipation*: by bland soft diet, mild laxatives and regular habits.

2-*Topical agent (ointment or suppository):* local anaesthetic, decongestant and antiseptic.

3-Baths: warm, after motions, for 15 minutes.

N.B. Recently, botulinum toxin injection in sphincter is tried as chemical denervator.

II- Surgical ttt: For chronic fissure and resistant acute ones:

1- Forceful dilatation under general anaesthesia to relax sphincter.

2- Lateral internal sphincterotomy: Division of the internal sphincter away from the fissure.

3-Dorsal fissurectomy and sphincterotomy: Triangular excision of the fissure and sentinel pile together with division of lower fibers of internal sphincter.

Hemorrhoids = Piles

Definition:

They are varicosity of veins in relation to the anus (piles = balls)

I- External piles: Varicosities of ext. haemorroidal plexus.

II- Internal piles: Varicosities of int. haemorroidal plexus = tributaries of the sup. rectal vein

Internal haemorroids

Aetiology:

I-Primary internal piles:

-It may be due to hereditary weakness of the wall of veins.

The anatomical predispositions are:-

1-The veins lie in the loose submucous C.T of the anorectum (not supported).

2-The veins are valveless (portal system is valveless).

3-The veins pass through the musculosa, which obstructs the flow during its contraction in defecation especially in constipation.

II-Secondary internal piles: (obstruction to venous flow):

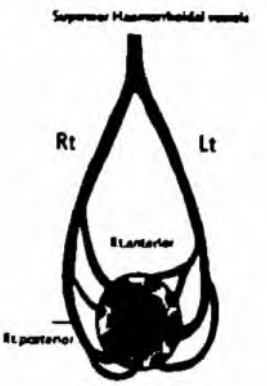
a) *At the level of the heart*: congestive heart failure.

b) *At the level of portal vein:* portal hypertension (rare).

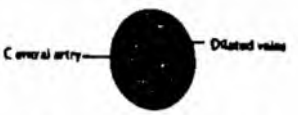
c) *At the level of the pelvis*: pregnant uterus, pelvic mass.

d) *At the level of the rectum itself*: CA. rectum, Ch. constipation.

Pathology



Site of piles


Cross section of a pile

Site:

-Internal hemorrhoids are usually arranged in three positions.at 3, 7 & 11 O'clock in the Lithotomy position.

-Also, there are four daughter & one granddaughter piles.

Composition:

-Each internal pile is composed of punches of dilated veins with central arteriole which is a terminal branch of the sup. rectal art. embedded in loose C.T and covered by mucosa.

-It has a pedicle, which lies at the anorectal ring and may be accompanied by external piles.

Complications:

1- Bleeding: is usually mild → anaemia. Rarely severe → shock.

2- Prolapse: of piles or rectal mucosa.

3-Thrombosis: in prolapsed piles → marked pain, swelling & tenderness.

4-Inflammation: infection of thrombosed pile may cause portal pyaemia.

5-Strangulation and gangrene: due to sphincteric spasm.

Incidence:

About 40% of populations have symptoms due to pile at some time in their life.

Clinical picture:

Symptoms:

1-Bleeding: The earliest symptom .Bright red blood at defecation.

2-Prolapse: Later symptom in the form of mass (es) that protrude from the anus with defecation

3-Pruritis: due to irritation by mucous discharge.

4-Pain only with complications e.g. thrombosis. Non-complicated piles are painless.

Signs:

1- Inspection: non-complicated piles are seen, but not felt.

1st degree piles: no prolapse, but only bleeding.

2nd degree piles: prolapse with straining but reduces spontaneously.

3rd degree piles: prolapse with straining but reduces manually.

4th degree piles: are permanently prolapsed.

2- PR examination:

To exclude cancer rectum.

Investigations:

Proctoscope: To diagnose 1st degree piles and exclude cancer rectum.

Treatment:

I- Conservative treatment:

Indications:

1- Secondary piles.

2- First degree primary piles.

3- Some selected case of thrombosed piles.

4- Inflammed piles.

It consists of:

1- Avoid constipation: bland high residue diet, mild laxatives, and regular bowel habits.

2- Topical agent: local anaesthetic, antiseptics, and decongestants.

3- Treatment of the cause in 2ry piles.

4 Strong antibiotics in inflammed piles.

II –Injection treatment (Sclerotherapy):

Indications:

1- Non complicated first & second degree piles.

2- Bleeding piles especially 2ry ones.

3- Recurrence after surgery.

4- Very old & unfit patients.

Aim:

To produce submucous fibrosis that obliterates vessels & holds the mucosa to the musculosa.

Procedure: "Gabriel's high submucous injection "

-Under proctoscopic examination, 3-5 ml of *phenol in almond oil* is injected submucously (extravascularly).

Complications:

Include ulceration, infection, prostatitis, stricture ...etc.

III – Rubber band ligation = *Barron's method.*

Indications: Non complicated 2nd degree piles. It is excellent method that can be used in outpatient's clinic.

IV- Cryosurgery:.

Idea: Using liquid nitrogen, coagulative necrosis of piles by extreme cold is achieved. Mucoid offensive discharge continues for weeks

V- Photo coagulation: Using infrared or Laser application. This method didn't get popularity.

VI- Lord's manual dilatation (not used nowadays).

Gentle gradual dilatation up to the four fingers of both hands. Cure is probably caused by induction of thrombosis & fibrosis. The risk of sphincteric rupture and incontinence is high.

VII – Surgery: = Haemorroidectomy.

Indications:

1- 3rd & 4th degree piles.

2- Recurrence after injections or other methods.

3- Associated pathology which needs surgery e.g. fissure.

Procedures:

Ligature & excision (Morgan and Milligan operation)

The only three main piles are dissected transfixed and excised starting usually with the right post one (at 7'Oclock) to avoid masking of the field by blood .We must leave adequate bridges of skin and mucous memberane to avoid stricture and anal stenosis.

Complications:

1-*Bleeding*: 1. Reactionary (within 24h) → ligation under general anaesthesia & pack.
2. Secondary (after 7-10 days) →blood transfusion, antibiotics, and pack around a rectal tube.

2-*Retention of urine*: due to reflex spasm of urinary sphincter→.

1- Hot fomentation. 2- Carbachol. 3- Catheter.

3-*Anal stenosis*: due to excessive removal of skin & mucosa.→

1. Repeated anal dilatation 2. V-Y anoplasty.

4-*Fissure*: usually responds to sphincterotomy.

5-*Anal incontinence*: due to injury of the sphincter.

II External piles

1- Thrombosed external hemorrhoids or perianal haematoma.

Cause: Straining or coughing causes rupture of anal venule of the ext. haemorroidal plexus → haematoma and thrombosis.

Clinical picture: painful bluish swelling in the perianal region of acute onset.

Treatment: excision under local anaesthesia.

2- External piles accompanying internal ones: = interno-external piles.

3- Sentinel piles: associated with ch. anal fissure.

Rectal Prolapse

Definition:

It is decent of the rectum through the anal canal either partially (mucosa only) or completely (full thickness of the wall = procid entia).

Partial prolapse

Complete prolapse

Actiology:

A- In children

1-Anatomical predisposition:

Straight course of the rectum due to still undeveloped sacral curve.

2- Increase in pushing force.

a- Prolonged straining e.g. gastroenteritis. & dysentery.

b- Chronic cough e.g. pertussis.

c- Bad toilet training (leaving the child for a long time on the pan).

3- Decreased supporting force.

Reduction of perirectal fat & weak pelvic floor e.g. malnutrition.

B- In adults.

1- Increase in the pushing force:

A- Straining from urethral obstruction or ch. constipation.

B- Increased intra-abdominal pressure e.g. pregnancy or pelvic tumours.

2-decrease in the supporting force:

A-After sphincterotomy operations.

B-Weak pelvic floor e.g. old age, obesity and neurological disorders.

C-Generalized mesodermal defect: e.g. visceroptosis.

3- Traction force: by large piles or polyps

Pathology:

Types: There are two types.

Item	Partial prolapse	Complete prolapse
1- incidence	Commoner	Less common
2- age	Common in children	Common in elderly
3- layers	Mucosa only	Full thickness of the rectal wall
4- length	Less than 2 inches	More than 2 inches
5- the mucosa	Shows longitudinal furrows	Shows circular folds
6- thickness	Thin (two layers of mucosa)	Thick (double rectal wall)
7- gurgling sensation	Absent.	may be present.

Complications:

1- Ulceration, Bleeding and infection.

2- Pruritis ani due to mucus discharge.

3- Irreducibility → strangulation →gangrene

4- Faecal incontinence: due to stretch of the sphincter & pudendal nerves

Incidence:

It is the disease of extremes of life, common in children (1-5years) and elderly

Clinical picture:

Symptoms:

1-Something protrudes from the anus at straining.

2-Symptoms of complications.

Signs:

Inspection: invites the prolapse by straining in squatting position.

Palpation: detect the thickness.

P.R: to detect the tone of the sphincter.

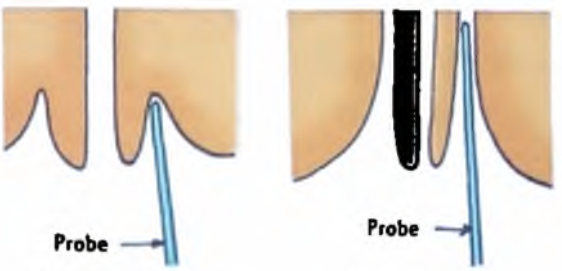
Investigations:

1-Anorectal manometry.

2-Sigmoidoscopy to exclude malignancy.

Differential diagnosis:

-From intussusception in infants:



Rectal prolapse Intussusception

In prolapse, there is no obstruction and the finger or probe can not be introduced in the groove between the mass and the anal margin.

Treatment:

I- In children

A-Partial prolapse:

1. Conservative ttt:

For mild non-complicated cases. *It consists of:*

a- Treat the cause, avoid constipation. & improve the general condition.

b- The mother is taught to reduce the prolapse and strap the buttock together.

c- Defecation in lateral position.

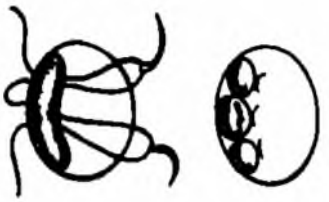
2-Submucous injection:

Injection of alcohol or phenol in almond oil (5%) to fix the mucosa to wall of the rectum.

3- Perianal circlage (Thiersch's operation).

-Encircling the anus with metal wire or non-absorbable suture in the substance of ext. anal sphincter and tied around the little finger.

-This is left for 2-3 months.



Goodsall's sutures

B- Complete prolapse:

1-Perirectal injection of sclerosant material (as before) to fix the rectum to the hollow of the sacrum.

II- In adults.

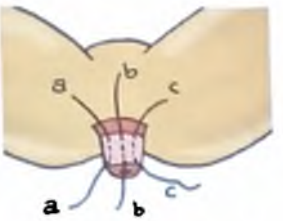
A-Partial prolapse.

1. *Submucous injection sclerotherapy.*

2-*Ligature excision* by *Goodsall's sutures.*

B- Complete prolapse.

Operations for complete prolapse can be performed through perineal or abd. approach.



Delorme's operation

I- Perineal approach:

1-*Thiersch circulage:* for elderly cases, unfit for major procedures.

2-*Mikculicz operation:* (Rectosigmoidectomy) .Recurrence is 50%.

3-*Delorme's operation:*

Remove the mucosal cuff of the prolapsed part then imbricate the musculosa with series of sutures then suture the two cut edges of the mucosa.

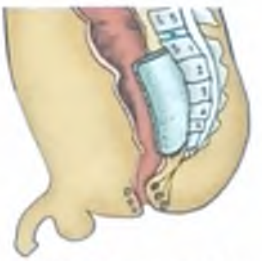
Lahaut's operation

4- *Lockhart Mummary's operation.* (perineal rectopexy).

II Abdominal approach:

1- *Lahaut's operation* :(abd. Colopexy).

Exteriorization of the sigmoid colon through the post. rectus sheath



Well's operation

2. *Well's operation* (abdominal. rectopexy):

Mobilization of the rectum then a sheet of polyvinyl alcohol (Ivalon) sponge is inserted between rectum and sacrum to induce fibrosis and fixation.

3.*Repstein operation:*

Hanging the rectosigmoid junction to the sacral promontory by a Teflon sling

4- *Graham 's operation* (abd. repair) : Prolapse is dealt with as a hernia of the rectovesical or rectovaginal pouch. (Excision of the sac + repair of the pelvic floor).

Anorectal Suppuration

I-Anorectal abscesses

Definition:

They are abscesses in relation to anal canal and rectum.

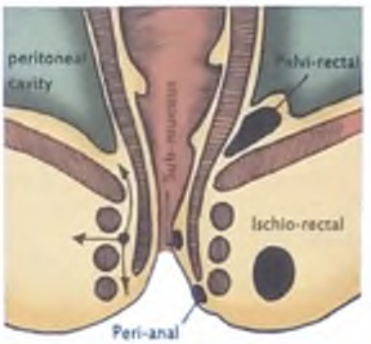
Aetiology:

I- Causative Organisms:

E. coli, staph. aureus , strept. , Cl. Welchii, Bacteroids, & Proteus

II- Route and mode of infection

In most cases, infection starts in the anal glands present in the intersphincteric space from which infection spreads to other spaces (see below)



Anorectal Abscesses

Pathology:

Sites & Types:

1- Perianal abscess (60%):

When infection spreads downward to the perianal space.

2- Ischiorectal abscess (30%):

-When infection spreads laterally through the ext. sphincter to the ischiorectal space.

3- Submucous abscess (5%):

When infection spreads medially through the int. sphincter to the submucosa.

4- Pelvirectal abscess (5%):

When infection spreads upwards above the levator ani muscle beneath the pelvic peritoneum. It usually arises secondary to appendicitis, parametritis, Crohn's disease etc.

Complications:

About two thirds of cases will develop fistula in ano due to:

1. Abundant fatty tissue in the ischiorectal fossae with poor vascularity.
2. The anal sphincter interferes with good drainage of the abscess.
3. Continuous contamination from the anal canal & perineum.

Clinical picture:

I- General manifestations or constitutional manifestations:

- Fever, headache, anorexia, & malaise.
- It is marked in ischiorectal & pelvirectal abscesses.

II- Local manifestations, *(dolor, tumour, calor, rubor and function laesa)*:

1. *Pain*: Throbbing, severe, and increased with coughing & sneezing.

2- *Hotness, redness & oedema:*

In perianal and ischiorectal abscesses (subcutaneous).

3. *Tender swelling*: according to the site.

A- Perianal abscess: small rounded swelling at the anal verge.

B- Ischiorectal: marked induration of the fossa better felt bidigitally.

C- Submucous: small swelling above the dentate line by PR exam.

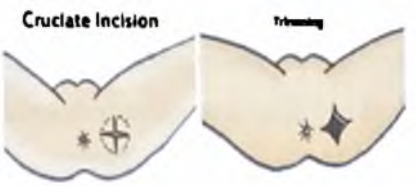
D- Pelvirectal: a large induration high up in the pelvis by PR or PV.

Treatment:

Don't wait for fluctuation = early drainage under general anaesthesia.

1,2) Perianal and ischiorectal abscesses:

Wide drainage through a cruciate incision with trimming of skin edges & deroofing of the abscess.



Drainage of ischiorectal abscesses

3) Submucous abscess:

It is drained with diathermy knife through a proctoscope.

4) Pelvirectal abscess:

It is drained through the ischiorectal fossa. The incision is deepened with division of the levator ani fibres to allow good drainage.

N.B. We don't wait for fluctuation in:

6 Ps	2Is	2Ss
1-2 Perianal & Perinephric abscesses	1- Intramammary abscess	1- Subphrenic abscess
3-4 Pulp & Palmer space infection	2- Internal organ suppuration	2- Submanibular cellulitis
5-6 Parotid & Prostate abscesses		Ludwig's angina

II- Anorectal Fistulas (Fistula in ano)

Definition:

An abnormal track communicating the anal canal or rarely the rectum with the perianal skin.

Aetiology:

-It always results as a complication of an anorectal abscess, which is drained spontaneously or by an inadequate surgical technique.

-Rarely, specific pathology e.g. Crohn's disease, colloid carcinoma of the rectum, are acused.

Pathology:

-The fistula is a track lined with granulation tissue .It has an internal opening usually at the anal crypt and external opening in the perianal skin.

-*Goodsall's rule*: Fistulas with an external opening in relation to the ant. half of the anus tend to be direct fistula and those with external openings in relation to the post. half of the anus (commoner) usually have curved track may be with multiple ext. openings and single internal opening in the posterior midline. long anterior fistula is an exception to the rule

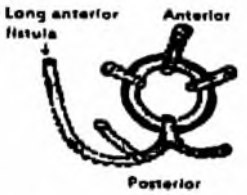
-Types (classifications)

I-Standard classification: there are 5 types (see the figure)

1. Subcutaneous. *2. Submucous.*

3. Low anal fistula. *4.high anal fistula.*

5. Pelvirectal fistula.



Goodsall's rule

II-Alan Parks classifications (see the figure)

1-Intersphincteric type (70%)

2-Trans-sphincteric (25 %).

3-Supra sphincteric (4%).

4-Extrasphincteric (1%).

Standard classification

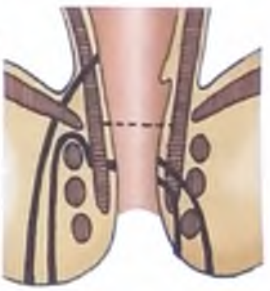
-Apart from the type of classification, the most important factor is whether the fistula opens below or above the anorectal ring.

1-Low anal fistula: opens below the anorectal ring

2-High anal fistula: opens above the anorectal ring

Complications:

1. Pruritis ani due to continuous purulent discharge.
2. Recollection & abscess formation when the ext. opening is blocked. The new abscess discharges through the same or new opening.
3. Colloid carcinoma may complicate long-standing cases.



Parks classifications

Incidence:

About two thirds of ano-rectal abscesses are complicated by fistula.

Clinical picture

I- Symptoms:

-History of anorectal abscess, that opens spontaneously or surgically drained but did not close.

-Typically, the patient complains of an opening near the anus discharging purulent discharge causing pruritis and soling of the under clothes.

II- Signs.

1- Inspection.

External opening: is a small hole in the perianal skin. It may extrude pus on pressure.

2- PR and bidigital exam:

The fistula is felt as a firm indurated track.

The internal opening may be felt as indurated depression Its relation to the anorectal ring should be carefully noted

3- Probing:

Should be done under anaesthesia in the operating theater.

Investigations:

1- *Fistulography*: not recommended nowadays except in difficult and recurrent cases as it may flourish infection.

2- *Proctoscopy*: to exclude carcinoma & detected int. opening.

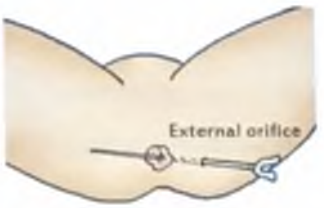
Treatment: = Surgery = fistulotomy or fistulectomy.

According to the type of fistula.

I- Intersphincteric and low trans-sphincteric fistula:

One stage fistulectomy.

Probing is done gently till it emerges from the int. opening ,then incision is done over the probe (fistulotomy). Trimming of the edge of the wound with *Salmon's backcut* to make the cutaneous part of the wound at least double the mucosal part (fistulectomy). The wound is left to heal from the bottom.



Probing

Fistulectomy

II- The high trans-sphincteric & suprasphincteric fistula:

Two-stage fistulectomy: protective colostomy may be needed.

First stage: is fistulectomy below the deep external sphincter with passing a stout silk suture round the deep part of ext. anal sphincter through the int. orifice to excite fibrosis that prevent wide gaping in the 2nd stage.

Second stage: 2-3 weeks later, excision of the remaining part of the track without risk of incontinence.

III- High fistulas (above the ano-rectal ring) = extra sphincteric fistula

Fistulectomy is contra indicated as it results invariably in incontinence due to division of the anorectal ring. The treatment entails:

1- Protective colostomy.

2-Conical drainage: cruciate incision of ischiorectal fossa and levator ani muscle to allow free drainage.

3- Treatment of the cause e.g. Crohn's ulcerative colitis.... etc.

Pilonidal Sinus

Definition:

It is a hair-containing sinus.

Aetiology:

I-Congenital theory:

It suggests that it arises as a result of infection of postnatal dermoid cyst.

II- Acquired theory: (accepted).

The skin is penetrated by short stout hair → Ch. irritation & 2ry infection. → Sinus formation.

It is accepted now due to :

1. It is much commoner in hirsute, black haired, adult males.
2. It is an occupational disease which occurs in the sacrococcygeal region in Jeep riders and in the inter-digital clefts in hairdressers.
3. It may occur in other hairy regions e.g. axilla, umbilicus.
4. No hair follicles were detected in the wall of the sinus and the hair projecting from it is dead hair projecting inwards.
5. The age incidence is between 20-30 years.

Pathology:

Site: The commonest site is the sacro-coccygeal region.

-It may rarely occur in the axilla, umbilicus, and digital clefts.

-The sinus extends in the subcutaneous tissue lined with granulation tissue. with frequent side tracks. In typical cases, hair tuft projects from the mouth of the sinus.

Sacro-coccygeal pilonidal sinus

Complications:

1-Repeated abscess formation.

2-Extension with sidetracks and may be with multiple openings.

3-Recurrence.

Incidence:

It is common in hirsute, black haired, obese adult males especially jeep drivers.

It is common between 20-30 y.

Clinical picture:

Symptoms:

1-Typically, the patient complains of abscess in the sacro-coccygeal region followed by a sinus with blood stained seropurulent discharge.

2- Recurrent abscesses with general & local manifestations.

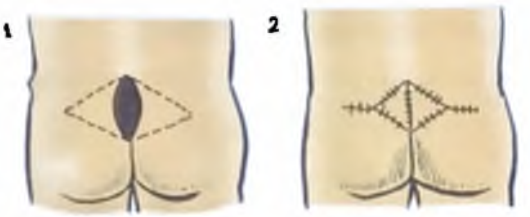
Signs:

1- Inspection:

- There is an opening in the midline of the back 2-3 cm above the anus discharging sero-purulent discharge. In typical cases, there is projecting tuft of hair.
- Occasionally there are multiple openings.

2- Probing:

The sinus passes upward and deep towards the sacrum but ends blindly short out of bone.



Double V-Y flaps

Treatment:

I- Abscess:

Drainage then treated as a sinus.

II-Sinus: → surgical excision:

1- Excision and suturing:

Wide excision of the sinus with all side tracks by elliptical incision down to the bone, and the wound is closed by interrupted sutures. In extensive sinus formation, skin flaps may be needed (rotational rhomboid, Z plasty or double V-Y flaps).

Z-plasty

Rhomboid flap

2- Excision alone:

Wide excision and leaving the wound open to heal by granulation from the bottom. Recurrence is rare but convalescence is long.

3- Marsupilization:

Lay open all tracks, remove all debris & hair then suture the edges to the skin. This procedure gives good, hairless scar with very low recurrence.

Cancer rectum & anal canal

Anorectal carcinoma

Actiology:

I-Premalignant lesions:

1-Adenomatous and villous polypi.
2-Familial polyposis and Gardener's syndrome.
3-Ulcerative proctocolitis and Crohn's disease.
} Like cancer colon

4-Fistula in ano may be complicated by squamous cell or mucoid carcinoma.

5-Simple papilloma of the anal canal.

6-Radiation dermatitis of the anal canal.

II-Predisposing factors:

Like cancer colon.

Pathology:

-Site

Upper 1/3: 35% Middle 1/3: 30% Lower 1/3: 30%

Multiple (multicentric) tumours are encountered in about 5% of cases.

-Macroscopic picture:

1-*Ulcerative type*: (mention criteria). It is common in the ampulla and anal canal.

2-*Cauliflower type*: irregular polypoidal mass, commoner in the ampulla.

3-Annular: irregular stenosing growth, common in the rectosigmoid junction.

-Microscopic picture:

1-*Adenocarcinoma* with its different types:

a) Columnar cell adenocarcinoma.

b) Spheroid cell adenocarcinoma e.g. schirrous, encephaloid & anaplastic.

c) Mucoid (colloid) adenocarcinoma.

2- *Carcinoid tumour of the rectum.*

3- *Squamous cell & basal cell carcinoma & malignant melanoma*, in the anal canal.

Spread:

I-Direct spread:

1-*Intrinsic:* It occurs more in the transverse direction (1/4 circumference in 6 months).

2-Extrinsic: late.

a) Anteriorly: urinary bladder, & prostate in males. Vagina, uterus & ureters in females.

b) Posteriorly: sacrum and sacral plexus in both sexes.

II- Lymphatic spread: (see the anatomy).

1-*Zone of upward extension*: from the upper 2/3 of the rectum, lymphatics pass through the pararectal LNs to the superior rectal and inferior mesentric LNs (it is the main pathway).

2-*Zone of lateral extension*: from the lower 1/3 of the rectum (4cm), and the upper 2/3 of the anal canal (above the dentate line), lymphatics pass through the pararectal glands to the middle rectal and internal iliac LNs.

3-*Zone of downward extension* : from the anal canal below the dentate line, spread occurs to the superficial inguinal LNs.

-The pre-aortic and para-aortic L.N.s are involved very late.

III- *Blood spread:* (late):

Mainly to liver, lung and adrenals.

IV- *Peritoneal dissemination:*

Only in advanced tumour of the upper 2/3.

Duke's A

Staging: (*Duke's classification*):-

Stage A: -The growth is confined to the rectal wall (5%).

-The prognosis is excellent.

Stage B: -The growth spread to the extra-rectal tissues, but no regional lymph nodes are involved (25%). (Good prognosis).

Duke's B

Stage C: There are 2ry deposits in the regional L.N.s. (50%).

-C1: the pararectal L.N.s are only involved.

-C2: the inf. mesentric LNs are involved (bad prognosis).

Stage D: -Distant metastasis (Inoperable) (grave prognosis).

Duke's C1

Complications:

1-Acute on top of ch intestinal obstruction.

2-Infection, ulceration & bleeding per rectum.

3-Secondary piles & ano-rectal suppuration.

4-Local & distant spread with their complications.

Incidence

-It is one of the commonest malignancies in the world.

-It is more in old (> 50y) males.

Duke's C2

Clinical picture:-

I- Symptoms:

1-*Bleeding per rectum*: is the commonest presentation.

2- Progressive constipation especially in carcinoma of the upper one third. Spurious diarrhea may result from accumulated mucous or use of purgative.

3-*Tenesmus:* It is a very common symptom especially in cancer lower 1/3.

4-*Pain*: is usually late due to: a-infiltration of the anal canal (somatic innervastion).

b-infiltration of sacral plexus.

c-Infection e.g. Anorectal abscesses .

5-*Symptoms of complications*..

II- Signs:

1-General:

-Weight loss and cachexia are late.

-Abdominal distension due to ch. obstruction or ascites.

-Enlarged Virchow's or inguinal LNs & hepatomegaly (metastases).

2-PR exam:

-It can detect about 90% of cases.

- It determines the site, size, extent (Pararectal L.N.s may be felt) & mobility of the tumour.

Investigations:

1-Proctosigmoidoscopy:

A-it can show the growth in almost all cases.

B-Biopsy should be taken for histopathological examination.

2--Barium enema:

When multicentric lesions or multiple polyps are suspected.

3- S. carcinoembryonic antigen (CEA).

Proctosigmoidoscopy

Differential diagnosis:

1-From benign strictures e.g. ulcerative colitis, & diverticulosis.

2-From other causes of tenesmus e.g. amoebic colitis.

Treatment:

A-Non obstructed cases

I-Operable cases: = Radical resection.

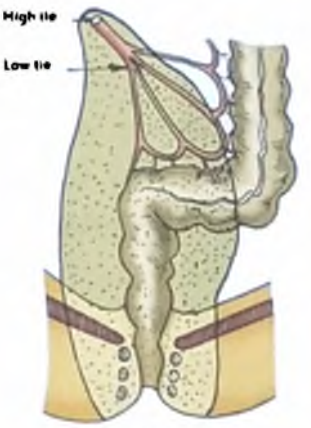
One of the following procedures:

1-Abdominoperineal resection of Miles: (The most popular).

-We remove:

1-Lower 3/4 of the sigmoid and its mesocolon.

2-The rectum and para rectal tissues.

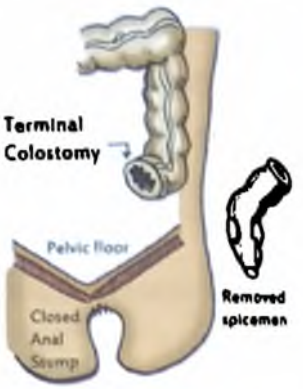


Radical resection

3-The anal canal and much of the perianal tissues & skin.

4-Part of the pelvic floor and its covering peritoneum.

-The operation ends by permanent left iliac colostomy .



Hartmann operation

2-Perineoabdominal resection of Gabriel:

-It is actually abdomino- perineo-abdominal i.e. (Abdominal exploration, Perineal resection then abdominal resection).

-It is more difficult & time consuming.

3-Synchronous (combined) resection:

Two surgical teams operate synchronously to shorten the operative time and minimize shock.

4--Abdominal resection of Hartmann:

-For tumours in the upper one third (> 10 cm from the anus).

-He performed radical resection of the rectum through abdominal approach as usual then closed the anal stump below the pelvic floor and draws the colon as terminal colostomy.

5-Anterior resection of Dixon (Sphincter saving operation):

-It is applied for growths situated > 10 cm from the anus and are well differentiated.

-Resection is done as in Hartman procedure, bu, anal stump is anastomosed to the pelvic colon better with EEA stapler.

EEA stapler

6-Pelvic exentration (Burnschwig's operation):

-Total pelvic exentration when the tumour has infiltrated other pelvic organs but is still operable.

-Posterior pelvic exentration is usually sufficient in females.

II- Inoperable cases:

1-Palliative resection.

2-Radiotherapy: adjuvant: preoperative, post operative or alone in unfit patients.

3-Chemotherapy: using 5-Flurouracil.

4-Immunotherapy: using monoclonal antibodies to CEA conjugated to cytotoxic agent.

B-Obstructed Cases

1-Resuscitation:

- IV fluids. - N/G suction. - Blood transfusion...etc.

2-Immediate laparotomy:

1) Resectable tumours:

A-Fit patient: abdomino perineal or Hartmamn's operation.

B-Unfit patient: sigmoid colostomy then radical resection later on.

2)-Irresectable: palliative sigmoid colostomy.

Prognosis:

It is comparable to that of cancer colon but less favorable.

Bleeding Per Rectum

Definition:

-Passage of blood per rectum.

-The term includes both Haematochezia and Melaena.

Haematochezia: is the passage of fresh blood per rectum.

Melaena: is the passage of altered blood (black, sticky, offensive) by digestive enzymes per rectum. It results from bleeding proximal to the ileocaecal valve.

Aetiology:

I- General Causes:

Bleeding tendency, hypertension ...etc.

II - Local causes:

(Any cause of bleeding from oesophagus to anal canal):

1-Oesophagus: Varices, ulcers and cancer.

2-Stomach and duodenum: Varices, peptic ulcers, and cancers.

3-Small intestines: Meckel's diverticulum, typhoid enteritis, TB enteritis & Crohn's disease.

4-Colon & rectum: - bilharziasis, angiomatous malformations, polyps, cancer, diverticulosis coli, ulcerative colitis, haemangiomas, and intussusception.

5-Anal canal: - piles, fissure, and cancer.

N.B. The commonest cause is bleeding piles.

Diagnosis:

A- History

1-Age of patient:

A-Infancy, The commonest is intussusception.

B-Childhood: commonest is rectal polyp (juvenile).

C-Young adult: commonest is piles.

D-Elderly: think of cancer.

2-Type of bleeding:

It may give an idea about the site of bleeding as follow:

A-Bright red blood on the surface of stool or even apart from defaecation → from anal canal

b- Red coat around stool → rectum and left colon.

C-Fully mixed with stool → right colon.

D-Black, sticky, offensive stool (Melaena) → from causes proximal to the ileocaecal valve.

N.B. *massive bleeding from proximal GIT (1.5 liters of bl.) lost rapidly appears as fresh bleeding per rectum (Haematochezia)*

3-Associated symptoms:

A-Heart burn, dyspepsia, & haematemesis → peptic ulcer.

B-Abdominal colic, constipation, & abdominal mass → intussusception.

C-Profound shock, severe abd pain → mesentric vascular occlusion.

D-Cachexia, anorexia, anaemia → malignancy.

E-Tenesmus, mucous + bl in stool → proctitis e.g.

F-Bleeding and pain → anal fissure or cancer.

B-Examination:

1-General:

For anaemia and general manifestations of the cause.

2-Abdominal:

A-Epigastric tenderness→ peptic ulceration.

B-Abdominal causes: cancer, T.B, Crohn's, bilharziasis, intussusception.

C-Hepatosplenomegaly ,Ascites → esophageal varices

3-Anorectal examination:

To detect piles, fissure, polyps, cancer, prolapse ... etc.

C-Investigations:

1- Stool analysis: for occult blood, bilharziasis, amoebiasis, etc.

2- Colonoscopy.

3- Selective angiography.

4- *Barium meal or enema:* should be delayed after endoscopy and selective angiography.

6- Complete blood picture and coagulation & bleeding time.

Treatment:

- Resuscitation in shocked patients i.e. I.V. fluids, blood transfusion, etc.
- Treatment of the cause.

The Spleen

ANATOMY

Spleen is the largest single mass of lymphoid tissue (Lymph node of the blood) .It measures about 1 X 3 X 5 inches and weighs 7 ounces.

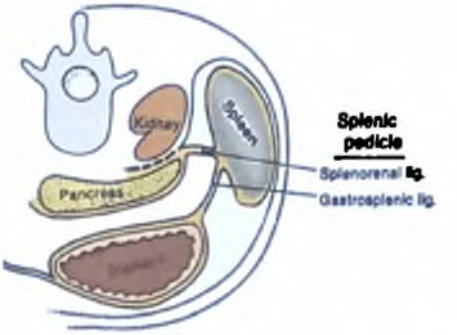
Position & surface anatomy:

-It lies in the (Lt. hypochondrium among the stomach diaphragm, left kidney and Lt. colic flexure.

-It extends from the 9th, to the 11th rib.

-Its medial end is 1.5 inches from 10th thoracic spine and its lateral end reaches the midaxillary line.

Borders, surfaces and relations:



-It has two borders:

*Anterior border, sharp and notched.

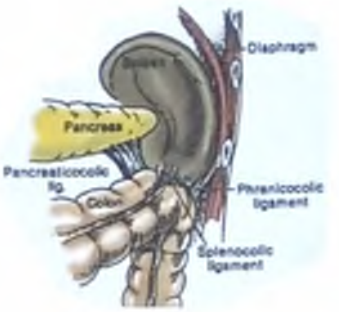
*Posterior (lower) border, thick and smooth.

-It has two surfaces:

*Ventral or visceral surface related to stomach, Lt. kidney, tail of pancreas, & Lt. colic flexure.

*Diaphragmatic surface related to the diaphragm separating it from the Lt. pleura and Lt. lung.

Ligaments of the spleen:

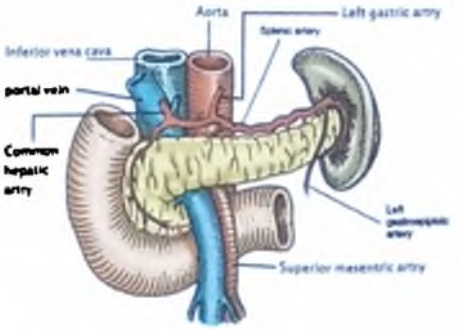


1-*Gastrosplenic ligament* connects the fundus of the stomach to the lat. edge of the hilum.

2-*Lienorenal ligament*, from the medial edge of the hilum to the ant. surface of the Lt. kidney.

3-*Phrenico-colic ligament*, stretches from the under surface of the diaphragm to the Lt. colic flexure in front of spleen

Blood supply:



1-*Arterial*: splenic artery from the coeliac trunk.

2-*Venous*: splenic vein → portal vein.

Nerve supply:

Branches from the coeliac plexus around the artery.

Lymphatic drainage:

To the pancreatico-splenic LNs → coeliac LNs.

Physiology

The spleen has the following functions

I- Haematological functions

1) Erythropoiesis : in the fetal and early years of life , the spleen can share in the production of red & white blood cells
2) Sequestration & destruction of old blood elements; aged and abnormal blood cells and their cellular debris are eliminated by spleen . Also spleen sequestrates normally up to 30 % of normal platelets. In huge splenomegaly up to 80 % of platelets are sequestrated in the spleen

II- Immunological functions

1) The spleen plays an important role in development of both B & T lymphocytes
2) Splenic macrophage can engulf and clear blood from bacteria, fungi, protozoa and foreign substance
3) The immunological cells of the spleen produce the following important immunological substances such as:
   a- IgM
   b- Opsonin, properdin and Tuftsin (important in phagocytosis)
   c- Interferon (antiviral agent)

I- Congenital anomalies

[1] Agenesis : absent spleen (rare) associated with immunological deficiency

[2] Accessory spleens (splenules) present in 10 – 35 % of population. The commonest sites are hilum of spleen, the greater omentum, along the splenic vessels over pancreas in this order of frequency

[3] Polysplenia : the spleen has deep furrows that divide the spleen into multiple masses

[4] Wandering (ectopic) spleen: the spleen has redundant mesentry that allow spleen to move widely in the abdomen even crossing the midline. Torsion is an imminent risk

[5] Congenital splenic cysts: dermoid or mesenchymal inclusion cysts

II- Trauma

Ruptured spleen

Aetiology:

I-Predisposing factors:

1-Splenomegaly: it comes out from the rib cover.

2-Pathological spleen: It is more friable e.g. malarial spleen, & septicemia.

II-Precipitating factors:

1-Direct trauma: - Closed: run-over and crushing injury.

- Open: stab, firearm & operative trauma.

2-Indirect trauma: acceleration - deceleration injury e.g. fall from a height.

3-Spontaneous rupture: unnoticed trauma to a pathological spleen (Malaria).

Pathology: -

Types: -1) Subcapsular haematoma.

2) Superficial tear (s).

3) Deep tear (s) reaching the hilum of the spleen.

4) Avulsion of one pole.

5) Complete depulping.

6) Avulsion or injury of the vascular pedicle.

Types of injury

Complications:

1-Shock: haemorrhagic & neurogenic.

2-Haemorrhage: - internal & may be external.

3-Concomitant injury to other organs e.g. Lt. kidney, diaphragm, lung - etc.

4-Late: infection → subphrenic abscess.

Incidence:

It is the commonest abdominal organ injured by blunt abdominal trauma

Clinical picture:

There are four clinical types:

1-Fatal type: -

Severe haemorrhage with rapid shock and death due to injury or avulsion to splenic vessels.

2-Classical type: pass into three stages:

A-Stage of shock: immediately after trauma, the patient faints and the spleen bleeds with developing shock e.g. rapid pulse, hypotension, rapid shallow breathing, & low temp.

B-Stage of recovery (Lucid interval): The bleeding stops and bl. pressure rises. The patient awakes and feels better and may go to the hospital or even to his work, but still there are:

1-Tachycardia.

2-Normal Bl. p., but there is orthostatic hypotension.

3-A wound or ecchymosis may be present in the Lt. hypochondrium.

4-Tenderness, guarding & even rigidity over the Lt. hypochondrium.

5-A click or crepitus of rib fracture may be detected.

C-Stage of internal haemorrhage:

When the blood pressure rises, the haemorrhage begins again with developing shock (increasing pallor, rising pulse rate, progressive hypotension ... etc).

On abdominal examination:

1-Generalized abdominal tenderness & rigidity maximum over left hypochondrium.

2-Shifting dullness in the flanks.

3-Balance's sign: shifting dullness on the right and fixed dullness on the Lt. side due to clotted haematoma around the spleen.

4-Kehr's sign: referred pain to the Lt shoulder induced by raising the foot of the bed due to irritation of the Lt copula & Lt phrenic nerve.

5-PR: exam. Tenderness & may be soft swelling.

3- Delayed type:

Like the classic type, but the stage of internal hage is delayed for days or even weeks due to:

1-Subcapsular haematoma that burst.

2-Small tear which was closed by omentum, or blood clot, which was dislodged.

Cullen's sign: bluish discoloration around the umbilicus. It occurs 1-2 days after trauma without surgical intervention due to diffusion of bl. around the falciform lig. to the umbilicus.

4-Spontaneous type:

Manifestations of internal haemorrhage develop, but without history of trauma.

Investigations:-

1-Plain X ray: of abdomen in erect position may show:-

a-Fracture Lt. lower ribs. b) Obliteration of the psoas shadow.

c-Raised Lt. copula of the diaphragm. d-Indentation of the Lt. side of the gastric air bubble.

e-Evidence of haemothorax or haemopneumothorax on the left side.

2-Abd U/S: It confirms internal haemorrhage and assesses its amount.

3-C.T with contrast: - The gold standard in haemodynamically stable trauma cases. It confirms splenic injury and defines its grade. Also, it diagnoses associated injuries of other viscera

4-Abdominal paracentesis or better Diagnostic Peritoneal Lavage (DPL) yielding bloody return

5-Selective splenic angiography & splenic scan in delayed type.

Treatment:

I- Hemodynamically stable patients can be conserved under ICU care with high success rate.

II- Haemodynamically unstable patients:

A- Resuscitation, correction of shock, bl. transfusion, and morphia.

B-Surgery: Immediate laparotomy then either:-

1-Splenectomy:

In children, immunization with polyvalent pneumococcal vaccine (pneumovax) is given to reduce the incidence of *overwhelming postsplenectomy sepsis* (see later).

2-Splenic preservation procedures:

Aim: to preserve as much viable splenic tissue as possible to preserve its immunological function and accordingly, prevent post splenectomy sepsis.

Procedures:

1-Splenorraphy: suturing with omental patch or flap.

2-Ligation of the splenic artery: may be done with splenorraphy.

3-Partial splenectomy.

4-Splenic auto-transplantation: slices of viable splenic tissue are wrapped by omentum and left to take a new blood supply

III- Inflammations

1) Acute splenic abscess: it rarely follows the course of certain fevers especially Typhoid or pyaemia from a distant septic focus. Treatment is splenectomy.

2) Chronic abscess it may follow neglected acute abscess or infection of splenic cyst, the treatment is splenectomy

3) Tuberculosis: it usually occurs in miliary TB. Multiple cold abscesses develop in the spleen

Hypersplenism

Definition

It is hyperfunction of the spleen to sequestrate and destroy blood elements (s)

Aetiology

I- Primary (idiopathic) hypersplenism : for unknown cause a normal spleen overdoses its destructive job

II- Secondary hypersplenism: hypersplenism complicating splenomegaly due to various causes especially portal hypertension

Pathology

As a result of over destruction of blood cells there will be pancytopenia (anaemia , leucopenia & thrombocytopenia) and there will be compensatory hyperactivity of bone marrow to

compensate for this high rate of destruction . The net result is the following triad splenomegaly , pancytopenia and overactive bone marrow

Clinical picture

Manifestations of the disease are the sequel of pancytopenia

1) Pallor , easy fatigability due to anaemia
2) Frequent infection especially sore throat , oral ulcerations , fevers due to neutropenia
3) Petichae & ecchymosis due to thrombocytopenia are very rare as there is compensatory hyperfunction of the platelets in spite of low number

Investigations

1) Blood count : reveals pancytopenia . one type may be more affected than the others , also reticlocytosis is usually evident
2) Bone marrow picture : it shows hypercellularity
3) Abdomenal U/S to diagnose splenomegaly and signs of portal hypertention

Treatment

1) Splenectomy usually corrects the haematological disorders. It may be accompanied by vasoligation or shunt operation in cases secondary to portal hypertention
2) Splenic artery embolisation : percutaneous transfemoral splenic artery embolisation causes splenic infarction and and subsequent atrophy with correction of haematological disorders . secondary infection , splenic abscess with septicaemia are serious complications that limit this line of treatment .

Splenomegaly

"The spleen should enlarges three times its normal size to be palpated abdominally"

To make a clinical diagnosis of Splenomegaly, you should find the following data:

-An intra-abdominal swelling in the Lt. hypochondrium .It may extend to the Lt. lumber or umbilical region and even to the iliac regions.

-Moves with respiration (except if fixed with adhesions). It moves downward and medially (to the right) with inspiration as the phrenco-colic ligament directs the spleen to the right

-It has a sharp anterior border with a notch (pathognomonic).

-The hand cannot be insinuated between it and the costal margin to get its upper pole.

-It does not show posterior (renal) ballotment.

-Its consistency is variable.

-It is dull on percussion & causing dullness in *Traub's area*.

A-Bacterial: septicemia, typhoid, T.B, splenic abscess, and syphilis.

B-Viral: infectious mononucleosis.

C-Parasitic: bilharziasis (*), malaria, Kala-Azar (visceral Leishmaniasis), Chaga's disease (Trypansomiasis), and hydatid disease.

II- Blood diseases:

A: Anaemias: All types e.g. thalassaemia major (*), spherocytosis ...etc.

(N.B. sickle cell anaemia may cause autosplenectomy due to repeated infarction).

B-Proliferative (*): leukemias, polycythaemia rubra vera, and myelofibrosis.

C-Autoimmune: Idiopathic thrombocytopenic purpura (I.T.P.).

III- Circulatory:

A-Portal hypertension (*): liver cirrhosis, & periportal fibrosis.

B-Splenic vein thrombosis: e.g. in cancer head of pancreas.

C- Splenic infarction.

IV- Metabolic:

A-Gaucher's disease (*): (glucocerbrosoidosis).

B-Amyloidosis (*): abnormal deposition of starch like substance in organs.

C-Porphyria: inborn error of haemoglobin biosynthesis.

V- Collagen diseases

A-*Still's disease*: juvenile rheumatoid arthritis, generalized lymphadenopathy & splenomegaly

B-*Felty's syndrome*: chronic rheumatoid arthritis & splenomegaly.

C-Systemic lupus erythematosis (SLE.).

VI- Neoplastic:

A-Benign: haemangioma.

B-Malignant: Hodgkin lymphoma, Non Hodgkin lymphomas (*) & primary fibrosarcoma.

C-Secondary malignant deposits (rare).

N.B. (*) refers to huge spleen.

Common causes of splenomegaly

1-Malaria

-Endemic countries e.g. China & India.

-It is usually huge spleen with sharp border.

-It is usually very soft, so ruptures on slightest trauma.

2-Spherocytosis

Definition:

It is a primary hemolytic anemia due to defect in the membrane of RBCs (autosomal dominant), leading to small bispherical RBCs that are fragile.

Aetiology:

The defect lies in the cell membrane, which makes it more permeable to Na^+. This stimulates the K^+/Na^+ pump to work faster and consume more energy. Early depletion of energy stores in RBCs occurs and leads to their premature senility and fragility. When RBCs pass through the spleen (stressful environment), they are sequestrated & rupture (haemolysis).

Pathology:

-Haemolysis ⟶ hemolytic jaundice (see before).

- Chronic anaemia ⟶ Extramedullary haemopiosis ⟶ Hepato-splenomegaly.
  Intramedullary haemopioesis ⟶ Reticulocytosis.

Complications:

1-Haemolytic crises: attacks of fever, severe anaemia, jaundice, and (haemoglobinuria).

2-Pigment stone, inspissated bile syndrome and obstructive jaundice.

3-Leg ulcers in long standing cases.

Incidence:

The disease usually presents very early in infancy. It is equal in both sexes.

Clinical picture:

* Pallor (anaemia), and lemon yellow tinge of sclera (jaundice).
* Hepato-splenomegaly.
* Picture of complications.

Investigations:

1-Blood picture: Anaemia, RBCs appear small with loss of central pallor + reticulocytosis.

2-Serum bilirubin: Increased indirect bilirubin (+ve indirect Van Denberg reaction).

3-Osmotic Fragility test: Increased osmotic fragility begins at 0.7 %, and completes at 0.45 %.

4-Measuring the life span of RBCs: labeling of the patient RBCs with radioactive chromium ($Cr.^{51}$) with daily scanning. Sequestration of RBCs in the spleen can then be studied.

5-Abdominal U/S to detect gallstones.

Treatment:

-Supportive treatment with bl. transfusion or packed RBCs especially in haemolytic crises.

-Splenectomy usually provides a good solution as it represents the slaughterhouse of RBCs.

-If gallstones are present, cholecystectomy can be done during or after splenectomy but not before because, any operative stress before splenectomy may precipitate haemolytic crises.

3-Idiopathic Thrombocytopenic Purpura (ITP)

Aetiology:-

It is an autoimmune disease due to development of antibodies against platelets.

Clinical picture: -

-There is purpuric eruption on skin with bleeding from mucous membranes

-Splenomegaly in about 25% of cases.

Investigations:

-Positive **Hess test**: prolonged bleeding time with normal clotting time.

-Low platelets count (< 60.000) (Normal 250.000 - 400.000 /mm). Megakaryocytes in bl. smear.

Treatment:

I- Medical: corticosteroids with or without IV immunoglobulin + platelets transfusion.

II- Surgical: splenectomy is indicated in:

1-Resistant cases not responding to steroid therapy for more than 6month.

2-Relapsing cases more than two times after steroids withdrawal.

4- Thalassaemia

See later

5-Leukaemias

-Spleen enlarges in both acute and chronic leukemias, and reaches a huge size in chronic myeloid leukemia where it reaches the Lt. iliac fossa. It may be pitting *(Mofty's sign)*.

-Clinical picture: There are anaemia, bleeding tendency and bony pain (tender sternum), Hepatomegaly, lymphadenopathy & leukaemic deposits elsewhere.

-Investigations Bl. picture & bone marrow picture are usually conclusive and obviate the need for LN. biopsy.

- *Treatment* by cytotoxic drugs. Splenectomy is indicated only in:

a) Hypersplenism b) prior to bone marrow transplantation

6-Lymphomas

See before

7-Egyptian Splenomegaly

It is a syndrome of:

1-Intestinal & liver bilharziasis.

2-Splenomegaly.

3-Anaemia, leucopenia and may be thrombocytopenia.

4-Irregular fever.

Aetiology:

-Causative organism: see intestinal B.

-The liver pathology is caused by deposition of bilharzial ova around the portal radicals causing periportal fibrosis.

-The splenomegaly is due to:

* Immunological reaction: in response to portal hyperplasia by bilharzial toxin (early).
* Congestive reaction: in response to portal hypertension (late).

Pathology:

1-Liver pathology: (periportal fibrosis) it is either:

a-Fine (diffuse) type:

Due to ova deposition in the fine portal tracts.

b-Coarse (peripheral type).

Due to heavy ova deposition in the large portal tracts.

c-Mixed type: (The commonest type) .

- It combines the features of both types.

2-Spleen:

MAC: is usually markedly enlarged, with thickened capsule and sometimes with thick adhesions to the surroundings. Patches of old infarction (perisplenitis) may occur.

MIC: congested red pulp with eosinophilic infiltration & enlarged lymphoid follicles.

3-Intestines:

Picture of bilharzial colitis (see before).

Complications:

1-Portal hypertension with all its complication.

2-Hypersplenism → Pancytopenia.

3-liver pathology may end in cholaemia.

Incidence:

-It is very common in Egypt especially in the Lower Egypt (Delta).

-It is commoner in young male farmers between 25 and 50 years.

Clinical Picture:

The disease passes into 5 phases:

1-Hepatomegaly:

-The patient may complain of Rt. hypochondrial pain, dyspepsia & dysentery.

- The liver is mildly enlarged, firm, smooth & the spleen just palpable.

2-Hepatosplenomegaly:

-The patient begins to complain of Lt. hypochondrial pain, asthenia, irregular fever.

- Hepatomegaly with marked splenomegaly.

3-Splenomegaly:

-Progressive deterioration of general health.

-The liver is shrunken, while the spleen is hugely enlarged & may reach the right iliac fossa.

4-Ascites.

- The patient complains of bad health. progressive abd. enlargement & bilateral LL. swelling .
- Cachexia, anaemia and clubbing of fingers.
- The liver is more shrunken.
- Ascites usually with L.L. oedema.

5-Cholaemia (liver cell failure).

-The patient begins to develop manifestations of LCF (jaundice, encephalopathy etc.).

-The first three stages (1, 2 & 3) are called *compensated splenomegaly*, while the last two stages (4 & 5) are called *decompensated splenomegaly.*

-The decompensation is either vascular decompensation (bleeding varices) or cellular decompensation (LCF).

Investigations:

See portal hypertension.

Differential diagnosis:

From other causes of splenomegaly.

Treatment:

I-Compensated splenomegaly (stages I, II & III):

A- Uncomplicated cases: Medical treatment

1-Diet: high glucose, protein & vitamins.

2-Drugs: antibilharzial drugs e.g. Praziquantel in single oral dose of 40 mg / kg.

3- Liver support.

B- Cases complicated with hypersplenism alone:

Splenectomy alone is sufficient.

C- Cases complicated with portal hypertension or oesophageal varices:

See portal hypertension.

II-Decompensated splenomegaly (stages IV & V):

1-Ascites: See portal hypertension.

2- Liver cell failure (cholaemia):

- protein free diet. High glucose intake.
- Lactulose: splits in the colon → Lactose → diarrhea → wash ammonia.
  → Lactic acid → low pH → inhibits ammonia-production.
- Neomycin and other intestinal antiseptics → inhibit ammonia-producing bacteria.
- I.V. infusion of arginine -sorbitol solution: arginine fixes ammonia to urea.

Portal hypertension

Definition:

Rise of the portal pressure (pressure in the portal vein) more than 20cm water (>15 mm Hg).

Normally, it is 10-15 cm H_2o (= 7-10mm Hg).

Aetiology:

I-Prehepatic (portal vein): (19%)

1-Congenital atresia or stenosis of portal vein.

2-Portal vein thrombosis: septicemia, portal pyaemia.

3-Splenic vein thrombosis: trauma or pancreatitis.

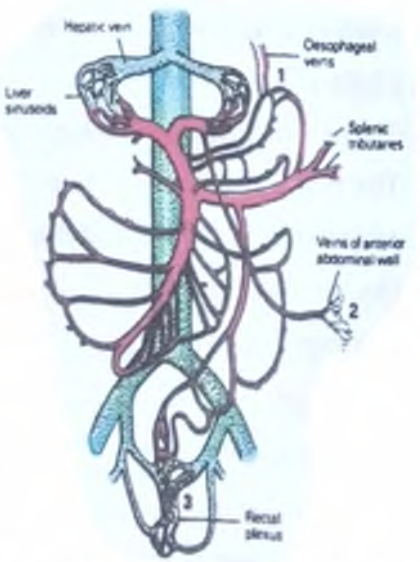
4-External compression: tumours e.g. cancer head

II-Hepatic: *(80%)*

1-Presinusoidal: periportal fibrosis (Bilharziasis).

2-Sinusoidal: liver cirrhosis.

3-Postsinusoidal: veno-occlusive disease (obstruction of small & medium- sized hepatic veins).



Causes of portal hypertension

III- Post hepatic *(1%)*

1-Budd Chiari syndrome (obstruction of main hepatic veins at their junction to I.V.C.).

2-Constrictive pericarditis.

3-Tricuspid stenosis or regurge & Rt. side H.F.

Pathology:

The rise in portal pressure has the following effects:

1-*Congestive splenomegaly* with secondary hypersplenism.

2-*Congestive gastroenteropathy*: leading to anorexia, dyspepsia & malabsorption.

3-*Dilatation of anastomotic channels between portal & systemic venous systems*:

Portosystemic anastomotic sites

Site	Portal side	Systemic side	Result
1-lower oesophagus	the Lt. & short gastric vv.	the azygous & hemiazygos vv.	Oesophageal varices
2-Anorectum	Superior rectal vein	Middle & inferior rectal veins	Piles
3-umbilicus	paraumbilical vein	Sup., inf. & superficial epigastric .vv.	Caput medusae
4-Bare area of liver	Portal capillaries	Rt. inf. phrenic vein	Silent
5-retroperitoneum	Sup. & inf. mesenteric v.v.	Veins of the post abdominal wall	Silent

Portosystemic collaterals act as safety valves that prevent much rise of the portal pressure. Therefore, they are beneficial, but oesophageal varices have a bad site.

4-*Hepatic dysfunction*: mainly due to the initial pathology but, deterioration of liver function occurs after bleeding and shock (ammoniacal encephalopathy).

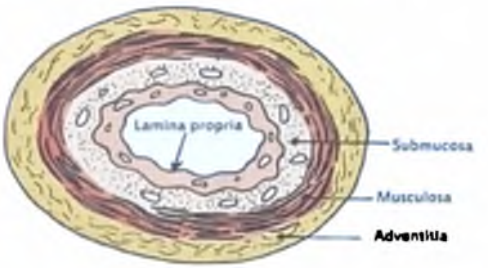
Complications:

1-Secondary hypersplenism: overactivity of the enlarged spleen to sequestrate & destroy blood elements. It results in a syndrome of pancytopenia, enlarged spleen & overactive bone marrow.

2- Malabsorption syndrome:

3-Bleeding varices:

The oesophageal varies are the most risky as they lie mainly in lamina propria and more liable to trauma. Bleeding occurs due to trauma of food, erosion by gastric acid or sudden increase in portal pressure e.g. straining.



Cut section of the lower oesophagus

4-Ascites: accumulation of free fluid in the peritoneal cavity due to:

a-Transudation of lymph from the liver surface (weeping liver) .

b-Hypoalbuminemia :due to decreased liver synthesis. It affects osmotic pressure.

c-Decreased hepatic metabolism of A.D.H. & aldosterone with salt & water retention.

d-Portal hypertension is merely a localizing factor.

Incidence:

Bilharziasis was the commonest cause in Egypt, nowadays, cirrhosis is the commonest..

Clinical Picture:

-History:

There may be positive history of bilharziasis or hepatitis.

-Symptoms: The onset is usually insidious:

-Anorexia, dyspepsia, and general weakness.

-Manifestations of complications may be the first presentation such as:

*Manifestations of hypersplenism: anaemia (weakness, pallor & easy fatigability) leucopenia (repeated infections), and thrombocytopenia (bleeding tendency).

*Bleeding varices: haematemesis and melaena.

*Manifestations of liver cell failure: poor health, jaundice, etc.

*Abd. distension due to splenomegaly and/ or ascites .

Signs:

-*General*: pallor, Jaundice, malnutrition, spider naevi, palmer erythema ... etc.

-*Abdominal:* -splenomegaly (mention characters).

-caput medusae.

-ascites.

Investigations: -

I- Investigations for the cause e.g., *bilharziasis . hepatitis ..etc):*

1- Stool & urine analysis to detect bilharzial ova + hatching test

2- Blood picture reveals oesinophilia, neutropnea & anaemia

3- Serological tests: ELISA, CFT, COPT

4- Sigmoidoscopy + biopsy from B. polyposis

5-Liver biopsy (by Chiba or true cut needle) (the surest method)

II-Investigations for liver function:

To exclude cirrhosis and evaluate fitness for operation (Child's classification).

III-Investigations for hypersplenism:

1-Bl. picture: pancytopenia.

2-Bone marrow picture: (sternal or iliac puncture): → active bone marrow.

3-Radioisotope scans: Injection of ^{51}Cr. tagged RBCs or ^{111}In labeled platelets to show the rate of splenic sequestration. Spleen: liver ratio > 2:1 is diagnostic.

IV-Investigations for portal hypertension:

A-Detection of oesophageal varices:

1-Barium swallow: multiple smooth filling defects in the lower oesophagus.

2-Oesophagoscopy: mandatory for detection and grading of varices.

-Grade I: Just elevation below the mucosa.

-Grade II: elevations & tortuousity with normal mucosa inbetween.

-Grade III: grade II with no normal mucosa in-between.

-Grade IV: In addition, there are dilated capillaries over the varices (varices upon varices) with erosions (red spots) which are risky sign (impending hage).

B- Visualization of the portal venous system and measuring the portal preasure:

1-Percutaneous spleno-portography and manometry.(obsolete)

2-Percutaneous trans hepatic portography and manometry (obsolete)

3- Hepatic venography by a balloon catheter in hepatic vein. By measuring the free (deflated balloon) & wedged (inflated balloon) pressure, portal pressure gradient can be calculated.

4-Indirectly, through the venous phase of splenic or superior mesenteric arteriography.

5-Digital subtraction angiography (DSA.).

6-Ultrasonography & CT.

7- MRI angiography can detect portosystemic collaterals and obstruction.

8-Duplex U/S gives full data about patency, amount & direction of flow in portal system. It is accurate and noninvasive.

9-Intra-operative portography: cannulation of a small vein in the lesser omentum then pressure measurement and injection of diodrast for portography.

Differential diagnosis:

1-Bleeding varices should be differentiated from other causes of upper GIT hage.

2-From other causes of splenomegaly, dyspepsia, & ascites.

Treatment

I-Uncomplicated cases:

A-Medical ttt: (liver support):

1-High glucose diet.

2-High protein diet (if liver function is not impaired)

3-Avoid alcohol & hepatotoxic drugs.

4-Beta blockers (propranolol) can be used as prophylaxis against variceal rupture.

B-*Interventional:* (prophylactic sclerotherapy).

It is recommended by some authors for silent risky varices (not accepted).

C-Surgery: (splenectomy + vasoligation)

It is not indicated except in grade IV varices (impending haemorrhage).

II-Complicated cases

A-Hypersplenism

Splenectomy + vasoligation.

B- Ascites:

1-Medical

a) Salt restriction + diuretics (spironolactone with or without lasix).

b) Correction of hypoalbuminaemia with or without I.V. infusion of salt free albumin.

c) Tapping of ascites: only if tense causing respiratory embarrassment.

2-Surgical: (obsolete)

a) *Talma Morrison op.* : (Omentopexy) : to drain ascetic fluid through lymphatics of abdominal wall (not done).

b) *Hepatic artery ligation:* to decrease lymphorrhea .It is dangerous and may cause acute LCF.

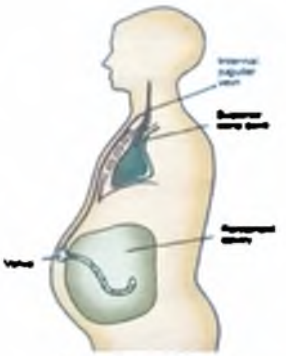
c) *Pleuro-peritoneal fistula*: May cause chest complications and usually ineffective.

d) Newmann's op.: Isolation of a loop of ilcum, which is left, opened in the peritoneal cavity to absorb ascetic fluid.

e) Saphenoperitoneal anastomosis: usually obstructed by kinking, thrombosis or omental plugging.

f) Peritoneal jugular shunt (Le Veen shunt & its Denver's modification): a synthetic one-way valve tube to shunt ascetic fluid from peritoneal cavity to jugular vein (most accepted).

Sapheno-peritoneal anastomosis



Le Veen shunt

The best treatment of resistant ascites is TIPS (see below)

C- Bleeding Varices:

I-During bleeding

1- Medical ttt:

1-Blood transfusion, fresh blood should be used.

2-IV. fluids, to restore blood volume till blood is available. Avoid NA^+ containing fluids.

3-Coagulants: vit K, Tranexemic acid.

4-Cimetidine: I.V. to decrease gastric acidity & avoid stress ulceration.

5-Sedation, avoid morphia & tranquilizers (metabolized by the liver).

6-Gastric lavage with ice-cold water, to induce vasoconstriction. It prevents encephalopathy, detect new bleeding and clear the stomach for endoscopic examination.

7-Vasopressin or better Glypressin (Terlipressin):

Dose: 20 units in 200cc glucose 5% over 20 min then, 0.4 u/min for up to 24 hours.

Action:

1- Causes splanchnic arteriolar constriction so, decreases portal pressure.

2-increases systemic pressure to combat the shock.

3-increases intestinal motility so helps evacuation of blood and reduces ammonia absorption

Side effects:

1-Abd. colic 2-Peripheral VC. →Raynaud's phenomenon 3-Coronary VC.→ angina

Contraindications:

Not used in patients over 50y, pregnant, angina prone with peripheral vascular disease.

N.B. glypressin has fewer side effects than vasopressin.

8-Somatostatin and its analogue Octreotide:

They have the same effects as vasopressin without significant side effect. They are now drugs of choice.

2-Acute endoscopic sclerotherapy:

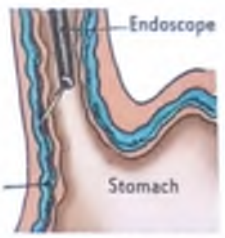
Substance: Ethanolamine oleate or sodium morrhuate (for oesophageal varices.). Histoacryl (Bucrylate) for gastric varices.

-*Technique*: Intravariceal injection. 1-3 ml in each varix.

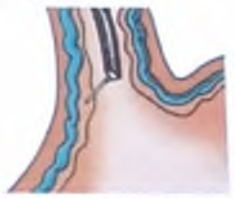
-*Aim*: to induce intravariceal thrombosis to stop bleeding.

-*Success rate*: Alone, it controls bleeding in 80-95% of patients.

N.B: The rebleeding is less with combined sclerotherapy and glypressin .Also; it is less with endoscopic rubber band ligation, which is similar to Barron's banding for piles.



Intravariceal injection sclerotherapy



Perivariceal injection sclerotherapy

3-Balloon tamponade:

Indication: when the bleeding continues inspite of sclerotherapy & glypressin or while preparing for sclerotherapy or surgery.

Types of tubes:

1-*Sungestaken Blackemore tube*: triple lumen. double balloon tube.

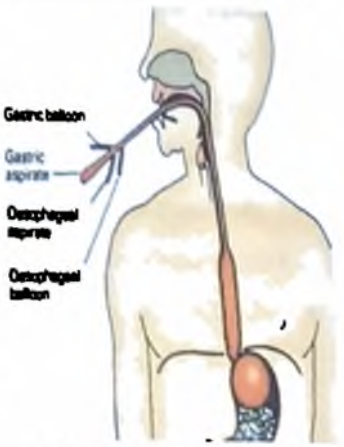
2-*Linton tube*: with single gastric balloon.

4-*Minnesota tube*: with additional fourth lumen to aspirate the oesophagus above the balloon.

Sungestaken -Blackmore tube

Technique:

1- Inflate the gastric balloon with 100-200 cc, then pull the tube to impact the balloon at the cardia to stop bleeding.
2- Inflate the oesophageal balloon up to 30-40 mmHg.
3- Gastric lavage is done through the gastric tube.
4- Oesophageal aspiration through the fourth tube to avoid aspiration pneumonia.
5- The balloon remains inflated for 12-24h while urgent investigations are done to evaluate fitness of the patient for operation according to *Child-Pugh classification.*



Minnesota tube in place

Item	A (1 point)	B (2 points)	C (3 points)
1-Ascites	Absent	Slight to moderate	Tense
2-Encephalopathy	None	Mild	Advanced (coma)
3-S. albumin	> 3.5 g/dl	3.5-3 g/d	< 3.0 g/dl
4-S. bilirubin	< 2.0 mg/dl	2.0-.3.0 mg/dl	> 3.0 mg/dl
5-International normalization ratio (INR)	< 1.7	1.7- 2.2	> 2.2

Class A (low risk) = 5-7 points with operative mortality 10%.

Class B (moderate risk) = 8-11 points with operative mortality 30%.

Class C (poor risk) = 12-15 points with operative mortality 80%.

The one year survival is 100%, 80% & 45% for class A, B, C respectively

Complications of Sungstaken's tube:

1-Aspiration pneumonia: The fourth tube in Minnesota tube can avoid this complication.

2-Suffocation if the gastric balloon ruptures. The oesophageal balloon ascends and causes asphyxia → urgent cutting of the tube.

3-Ulceration & necrosis of oesophageal mucosa: Do not let the tube more than 24 hours.

-The tube is deflated after 24 hours. If no bleeding recurs within 24 hours, remove it. If bleeding recurs, re-inflate it and prepare for:

*Urgent operation for class A & B.

* Interventional radiology e.g. Transhepatic variceal embolization (TVE) or *transjugular intrahepatic portosystemic shunt* (TIPS) for class C.

4- Interventional radiology

A- Transjugular Intrahepatic Portosystemic Shunt (TIPS.)

A recent method to stop variceal bleeding in patient with poor liver function using expandable metal stent.We create a shunt between intrahepatic portal radicals and hepatic veins inside the liver .

B- Transhepatic variceal embolization (TVE):

Under radiological guidance, gel foam is embolized into varices to stop hage.

TIPS

If bleeding continues inspite of the other measures → emergency operation.

5-Emergency Surgery

I- Disconnection (Non shunt) operations.

1-Boerema & Crile operation:

Trans-thoracic Trans-oesophageal variceal ligation.

2-Tanner's operation:

Ligation of the Lt. gastric & short gastric veins. with transection and reanastomosis of the upper stomach.

If splenectomy is, added→ modified Tanner operation.

Tanner's operation

3-Hassab's operation:

Splenectomy + ligation of the Lt. gastric & Lt. Gastroepiploic vessels. +Devascularization of the lower oesophagus.

Rifaat's modification He added perigastric omentopexy wrapping the omentum around the denuded stomach.

Hassab's operation

4-Segiura's operation:

Thoracoabdominal incision, splenectomy + devascularization of thoracic & abdominal oesophagus + oesophageal transection and resuture +pyloroplasty (due to inadvertent vagotomy).

* Transection & re-anastomosis of the oesophagus can be done now easily with EEA stapler introduced through a gastrotomy.

Transection with EEA

II-Shunt operations (obsolete)

A-Total (complete) portosystemic shunts

1-Portocaval shunt: (between portal vein & IVC).

Either end to side or side-to-side anastomosis.

2-Mesocaval shunt (between S.M.V. & I.V.C).

Either side to end or better H mesocaval shunt using venous graft or synthetic graft.

3-Central splenorenal shunt (between splenic & Lt. renal vein)

After splenectomy, the central stump of the splenic vein is anastomosed to the Lt. renal vein either ends to side.

Disadvantages of total shunts:

1-Portal blood enters the systemic circulation bypassing the liver (with high ammonia content not metabolized in the liver) → hepatic encephalopathy.

2-Decrease of the blood supplies to the liver → more deterioration of the liver function.

End to side Portocaval shunt

Side to side Portocaval shunt

H Mesocaval shunt

B-Selective decompression shunts

Aim: Isolation of the oesophagogastric area from the portal circulation and creating a new drainage to the systemic circulation to lower the venous pressure selectively in this area.

1- *Warren's operation = Distal splenorenal shunt (DSRS).*

Division of the splenic vein, ligation of the central stump and the distal one is anastomosed to the renal vein end to side. The Lt. gastric vein is ligated. It is not suitable for splenectomized patients or if the splenic is thrombosed.

2- *Lt. gastrocaval shunt (Inokushi operation).*

The Lt. gastric vein is anastomosed to the inferior vena cava by an autogenous saphenous graft together with splenectomy.

Central splenorenal shunt

Distal splenorenal shunt
Warren's operation

C-Stimulation of new porto-systemic collaterals

1- Omentopexy fixation of the greater omentum into the rectus sheath hoping to create new shunts with the veins of abd. wall.

2- Splenopneumopexy: transposition of the spleen in pleural cavity to create new shunts with pulmonary circulation.

Inokushi operation

II- After control of bleeding

a-Propranolol.

b-Chronic endoscopic sclerotherapy: (for child C):

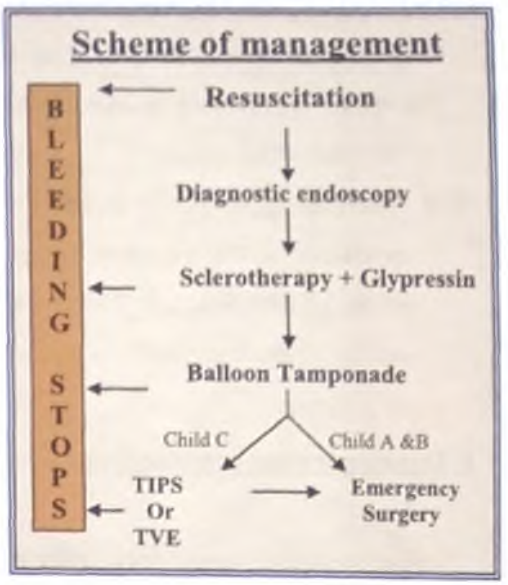
-Perivariceal injection: induces fibrosis that obliterates the oesophageal varices at 3 weeks intervals until all varices are obliterated.

c-Surgery: for child A & B :

-Splenectomy & vasoligation.

-Warren's operation (best) or H mesocaval for child A

-Liver transplantation: it is the only curative procedure and the most definitive treatment.



Prognosis:

-The mortality rate of bleeding varices is relatively high:

- 50% in the first attack - 70% in the second attack.

-The main prognostic factor is the liver condition, which determines the degree at which the body can withstand the stress of haemorrhage & shock.

Splenectomy

Indications:

I- Trauma (rupture spleen): the commonest indication.

II- Blood diseases: ITP, cong. haemolytic anaemia e.g. spherocytosis, and thalassaemia.

III- Portal hypertension:

1) Grade IV varices.

2) Bleeding oesophageal varices.

3) Hypersplenism.

IV- Primary hypersplenism.

V- Splenic abscess.

VI- Hodgkin's disease as staging laparotomy.

VII- As a part of other operations: e.g. gastrectomy and pancreatectomy .

VIII- Miscellaneous: torsion of the spleen, hydatid cyst& aneurysm of splenic artery.

Technique:

- Incision: Many incisions may be used most commonly Lt. paramedian or medline incision. Lt subcostal incision may be used in small splenomegaly

- The posterior layer of the leino-renal lig. is divided and the spleen is delivered into the wound
- The gastro-splenic lig. containing the short gastric vessels is clamped, divided & ligated.
- Then the pedicle is dealt with at the hilum taking care not to injure the pancreatic tail.
- The spleen is removed, haemostasis is secured and the wound is closed in layers with drains at the bed of the spleen.

N.B. Many surgeons prefer to begin with ligation of the splenic artery first to allow the blood contained in the spleen to enter the circulation. The lesser sac is opened through division of the gastro-colic lig. And the splenic artery is attacked at the gastric bed above the upper border of the pancreas

Complications:

I- Intra-operative complications:

1- Shock

2- Haemorrhage: commonly from tributaries of the splenic vein at the hilum of the spleen especially in portal hypertension.

3- Injury to important structures such as gastric fundus, pancreatic tail or splenic flexure of the colon

4- Difficulty: In cases with marked adhesions, delivery of the spleen is very difficult and risky as it may cause intractable bleeding. So, it is wise to leave the spleen and ligate the splenic artery in the gastric bed to reduce the size of the organ and minimize hypersplenism.

Postoperative complications:

1- Haemorrhage:

- It is usually reactionary due to
  - ❶ slipped ligature of the splenic pedicle (severe and usually fatal)
  - ❷ ooze from small vessels at the bed of the spleen especially in portal hypertension cases.
- It usually indicates re-exploration and dealing with the bleeder

2- Gastric injury:

Injury to the gastric wall during ligation of the short gastric vessels leads to haematemesis, gastric fistula or subphrenic collection.

3- Pancreatic injury:

Injury of the tail of pancreas during ligation of the splenic pedicle may precipitate acute pancreatitis

4- Colonic injury:

Injury to the splenic flexure of the colon may lead to colonic fistula or faecal peritonitis

5- Recurrence of hypersplenism:

It is due to enlargement of the splenculi (isolated splenic tissues) that may reach large size and continue to destroy blood elements. They should be searched for and removed.

6- Overwhelming post splenectomy infection (OPSI):

Removal of spleen especially in infants and children is associated with immunity defect against capsulated bacteria such as Pnumococci, H. infleunza & Meningococci that may induce a severe form of intractable sepsis with high mortality.

Preoperative immunoprophylaxis (vaccines) and postoperative chemoprphylaxis (antibiotics) should be given to children to avoid such serious complication

7- Portal vein thrombosis:

It may be due to extension of splenic vein thrombosis and/or post-splenectomy thrombocytosis. It may prove fatal.

8- General post operative complications (after major operations):

A- Pulmonary complications

❶ Basal atelectasis.

❷ bronchopnenmonia.

❸ lung injury

❹ pulmonary embolism secondary to DVT.

B- Acute gastric dilatation and paralytic ileus.

C- Burst abdomen

D- Infection usually in the form of the subphrenic abscess due to infection of infected haematoma at the splenic bed

THE LIVER

Anatomy

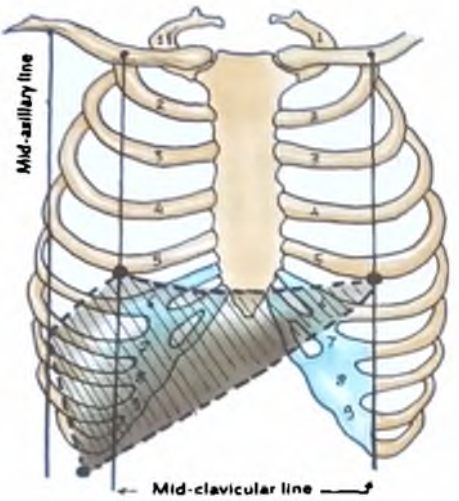
The liver is the largest parenchymtous organ and largest gland in the body

Surface anatomy (see the figure)

1- Upper border: from the Lt. 5th rib in the midclavicular line (MCL) to the Rt. 5th rib in the MCL. then to the Rt 7th rib in the midaxillary line

2- Right border: from the last rib to a point 1 cm below the Rt. costal margin

3- Inferior border: from the last point to the Lt. 8th costal cartilage then to the Lt. end of upper border



Surface anatomy of the liver

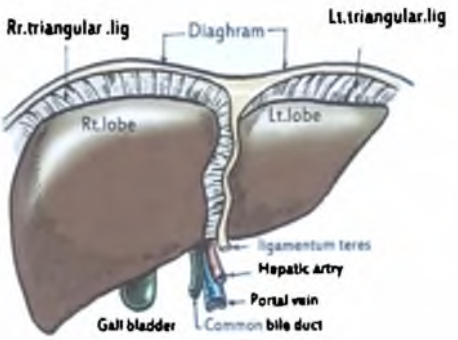
Ligaments of the liver

1- The upper & lower coronary ligaments & Rt. triangular lig. : between the Rt. lobe & diaphragm .

2- The Lt. triangular ligament: between the Lt. lobe & diaphragm.

3- The falciform lig. & ligamentum teres

4- Hepatoduodenal lig. (Lesser omentum).



Ligaments of the liver

Bare area (proper) of the liver:-

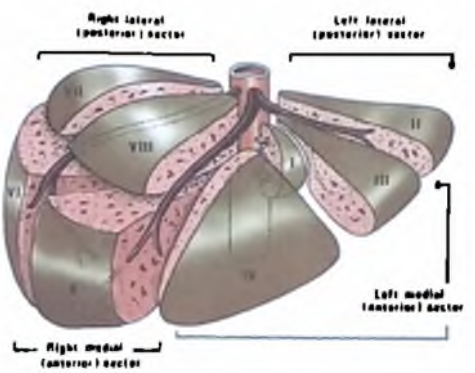
In the posterior surface of the Rt. lobe between the two coronary ligaments.

Liver segments:

-Morphologically, the liver has four lobes, right, left, caudate and quadrate lobes.

-From the surgical and anatomical point of view, the liver is composed of 8 segments according to segmental distribution of hepatic art., portal vein & common bile duct

-The main lobar fissure extends from IVC & gall bladder bed (see the figure).



Liver segments

Blood supply:

- The total hepatic blood flow is about 1.5 L/ min (25% of the cardiac output).
- The hepatic artery provides only 1/3 & the portal vein provides the other 2/3 of the flow. However, the liver gets 50% of its oxygen from the hepatic art. & 50% from the portal vein.
- Hepatic veins drain the liver into the IVC.

Nerve supply:

From the hepatic plexus at the porta hepatis containing both sympathetic (from coeliac plexus) and parasympathetic (from hepatic branch of the Lt. vagus).

Lymphatic drainage:

Lymphatics drain into hepatic group of LNs around hepatic artery then to the coeliac LNs.

I- Trauma

Liver Injuries

Aetiology:

As in splenic injuries.

Pathology:

Site: the right lobe is much commonly injured because it is larger & less mobile .The anterior or superior surfaces are usually the site of tears.

Types: as in splenic injuries (1-6) +

7- Injury or avulsion of extrahepatic biliary tract.

Complications: as in splenic injury. (1-4) +

5- Haemobilia: due to rupture of haematoma into the biliary tree.

6- Biliary leakage leading to biliary peritonitis, subphrenic abscess or biliary fistula

Incidence:

- It is the second commonest solid organ injured in blunt abdominal trauma
- It is the third commonest organ in firearm injury of the abdomen.

Clinical picture:

1- History of trauma to the upper abdomen or lower chest followed by shock & syncope.

2- Examination reveals :

- Wound or ecchymosis in the right hypochondrium.
- Tenderness & rigidity of the right hypochondrium then generalized.
- Crepitus of fractured rib may be elicited.
- Signs of internal haemorrhage (pallor, shock, generalized rigidity, shifting dullness etc.).

Investigations:

As in splenic injuries.

Treatment:

I- Haemodynamically stable patients can be managed conservatively with high success rate.

II- Haemodynamically unstable patients

1- Resuscitation: antishock measures: I.V. fluids, bl. transfusion, morphia - etc.

2- Operation: urgent exploration through abdominal or thoracoabdominal incision.

Pringle's maneuver: temporary occlusion of the hepatic artery and portal vein by application of a vascular clamp to the gastro hepatic omentum to control massive hage.

- Clean, straight tears should be sutured by mattress sutures over omental pad.
- Trauma associated with necrosis of some parts needs debridment or even partial hepatectomy.
- Injury to extrahepatic biliary passages needs biliary reconstruction.
- Drainage of the peritoneal cavity is mandatory to prevent biliary peritonitis.

Prognosis:

The mortality for hepatic injury is high (up to 20%).

II- Inflammation

Liver abscesses

Causative organisms:

1-*Bacterial:* - E. coli, Klebsiella, strept, staph., bacteroid → pyogenic abscess.

2- *Parasitic*: Endameba histolytic (commonest in Egypt)→ amoebic abscess.

B- Pyogenic abscess

Aetiology: Route of spread:

1- *Portal vein*: - pyaemic abscesses secondary to portal pyaemia.

2- *Bile track*: - following suppurative cholangitis → cholangitic abscess.

3- *Haematogenous abscess*: Bacteria reach the liver through the hepatic artery.

4- *Infection of a cyst or haematoma*.

Pyaemic liver abscess

Aetiology: It is now a rare condition. It occurs due to portal pyaemia secondary to obstructive appendicitis, colonic diverticulitis or infected haemmorroids.

Pathology: The liver is studded with multiple small abscesses.

Clinical picture: The patient is toxic with high fever and rigor. Jaundice commonly develops. The liver is enlarged and tender

Treatment: -Broad spectrum antibiotic, Bl. transfusion and IV fluids

- Drainage of abscesses if large and accessible either percutaneously or surgically.

C- Amoebic liver abscess

Aetiology:

Causative organism: Entameba histolytica.

Route of infection: Entameba is carried to the liver from the colon by the portal circulation.

The organism causes localized necrosis & liquefaction of the liver cells with abscess formation.

Site: commonly in the posterior part of the dome of the right lobe. (Why?)→Two theories:

1--Flow direction theory: the direction of flow in the portal vein points to the dome of the Rt. lobe.

2--The double stream theory: the bl. coming from the Rt. colon drains mainly to the Rt. branch of the portal vein. Entameba is concentrated in the right colon.

Number: solitary in 70 %

Size: variable

Wall : shaggy irregular

Contents: Anchovy sauce (chocolate pus)

Complications:

1- Secondary bacterial infection: Pyogenic abscess.

2- Rupture: it may rupture into

a) *Subphrenic space* → abscess

b) *Pleura cavity* :- empyema

c) *Lung* :- abscess or fistula

d) *Peritoneum* : peritonitis

e) *Bowel* : fistula

f) *Exterior* : pointing below ribs

3- Chronicity & calcification.

4- Liver cell failure.

Rupture of amoebic abscess

Incidence: -

It is more common in young adult males in tropical area (e.g. Egypt.).

Clinical picture: -

General manifestations (Toxaemia):

Fever, anorexia, malaise, loss of weight and may be tinge of jaundice.

Local manifestations: (of abscess):

1- Pain: dull aching in the right hypochondrium referred to the right shoulder.

2-Tenderness & rigidity: over the right lower ribs & right hypochondrium.

3-Enlarged tender liver.

Investigations:

A-Investigations to confirm amoebic aetiology

1- *Stool analysis, bl. picture, serological tests* (CFT & haemoagglutination test) most reliable.

2- *Sigmoidoscopy*: with or without biopsy. .

3- *Therapeutic test:* dramatic response to Emetine or metronidazole is characteristic.

B- Investigations to localize the abscess:

1-Plain X ray: shows elevated right copula of the diaphragm with pleural effusion.

2-*Ultrasonography, C.T scan, MRI & liver scan* (99Tcm sulfur colloid).

3-Diagnostic aspiration (sonar guided) reveals the characteristic chocolate pus.

Differential diagnosis:

From other causes of enlarged tender liver: hepatitis, pyogenic abscess, CHF & hepatoma.

Treatment

I-Conservative ttt:

1- Bed rest, good nutrition, vitamins & liver support.

2- Antiamoebic drugs: emetine hydrochloride, flagyl (metronidazole) or Tinidazole.

3- Antibiotics to prevent or treat 2ry infection.

II Aspiration:

Indications:

1- Failure of conservative measures for 5 days.

2- Very large abscess .

3- Very toxic patient.

Technique:

Under local anaesthesia and ultrasonic guide, a long needle is introduced through the 9th or 10th intercostal spaces in the right midaxillary line not more than 10 cm depth.

III Surgical (Open drainage):

Indications:

1- Failure of conservative measures and aspiration.

2- Secondary infection →pyogenic abscess.

3- Rupture of the abscess.

4- Abscess of the left lobe for fear of rupture into pericardium

Procedure:

Exploration, deroofing or incising the abscess and insertion of drain in it.

N.B.What are the compilations of open drainage?

1-Secondary hage.

2- Secondary infection.

3-Amoebiasis cutis: fatal: spreading cellulitis of the anterior abdominal wall caused by Entamoeba.

LIVER TUMOURS

A- Benign tumours

All benign tumours are rare

I- Tumour like conditions.

1- Haemangioma.

-It is the commonest variety. It is of the cavernous type.

-It is usually silent, discovered accidentally during laparotomy or investigations for other unrelated conditions, but it may rupture causing serious bleeding

Treatment.

-Asymptomatic cases need no treatment.

-Excision for relatively small tumours.

-Irradiation or feeding artery occlusion is resorted to in larger tumour.

2- Focal nodular hyperplasia (FNH).

- Harmless, idiopathic lesion of the liver.
- It may be related to parenchymtous injury.
- Microscopically, it resembles liver cirrhosis.

Treatment:

Most cases need no treatment.

Lesions suspicious of malignancy should be resected.

II- True neoplasms:

1- Liver cell adenoma (LCA).

- The old confusing name (benign hepatoma) is obsolete.
- It occurs almost only in females with a high incidence in those taking contraceptive pills.
- It forms a soft, yellow, solid mass, multiple in one third of cases.
- Microscopically, there are sheet of hepatocytes but without portal triad.
- It is usually asymptomatic but it may bleeds or change malignant.

Treatment

-Withdrawal of the pills is associated with a well-documented regression of the tumour.

- Suspicious tumours should be resected.

2- Cholangioma, & biliary cystadenoma.

- They are rare tumours from the bile duct epithelium.
- The latter reaches larger size with cystic degeneration.

B- Malignant tumours

I- Primary tumours.

Aetiology:

I- Predisposing factors.

1- Hepatitis B infection:, 90% of patients with hepatocellular carcinoma (HCC) patients are carriers of hepatitis virus. Hepatitis C virus seems to be more important than virus B

2- Race: HCC is common in Africa and cholangio-carcinoma is common in Chinese.

3- Aflatoxin ingestion: It is a toxin produced by the fungus aspirgillus flavus, which contaminate grains and flourish in warm, moist media. Bad storage of grains in Africa is predisposing factor.

4- Smoking & alcohol

5- Male gender and androgen therapy

II- Pre-malignant conditions :

1- Liver cirrhosis: post hepatitis cirrhosis and alcohol cirrhosis are the most premalignant

N.B. Bilharzial periportal fibrosis is not a premalignant condition.

2- Asiatic cholangitis: caused by clonorechis sinensis predisposes to cholangio carcinoma.

3- Sclerosing cholangitis: either primary or secondary

4- Benign tumours e.g. HCA.

5- Choledochal cyst and Caroli disease

Pathology:

Types:

1- Hepatocellular carcinoma (HCC).

MAC: Single or multiple (intra hepatic lymphatic spread) yellow masses

MIC: Malignant hepatocytes with acidophilic cytoplasm and large nuclei.

2- Fibrolamellar carcinoma.

-It is considered a massive subtype of HCC that occurs in non-cirrhotic young adults.

-There are numerous fibrous septa that divide the tumour into lobules

3- Cholangio carcinoma

-It arises from the epithelial lining of the biliary tree.

4- Hepato-cholangiocarcinoma

-A mixed tumour that combines histological features of both HCC & cholangio carcinoma.

5- Hepatic cystadeno-carcinoma.

-A large tumour formed of cystic spaces containing papillary projection.

6- Hepato blastoma

- A variety of HCC that occur in children. It contains embryonic liver cells besides bone, cartilage, striated muscles and connective tissue.

Complications:

1- Rupture with massive bleeding.

2- Obstructive jaundice: progressive painless obstructive jaundice if it obstructs bile ducts.

3- Cachexia and malignant ascites.

4- Paramalignant syndrome: e.g. hypercalcaemia, hypoglycaemia

5- Distant metastasis especially to the lung and bones.-.

Incidence:

- It is one of the commonest cancers in the world.
- HCC is common in Africa more in middle age males.
- Cholangio carcinoma is common in the Far East.

Clinical picture:

- Many tumours are discovered accidentally during abd. U/S done for other unrelated condition.
- A common presentation is deterioration of the general condition of a known cirrhotic patients the malignant triad (Athenia, Anorexia and Anaemia is usually present).
- Abd. examination usually reveals enlarged, tender, hard liver , with friction rub due to perihepatitis and arterial bruit due to high vascularity .
- Signs of complications e.g. Jaundice, malignant ascites.

Investigations:

1- Liver function test.

2- Tumour markers: Alfa fetoprotein is elevated in 1/3 of cases. Levels above 400 ng/ml is diagnostic of HCC (Normally it is 0-10 ng/ml) It is of good prognostic value.

3- Abd U/S, CT scan, MRI and MRA.

4- Hepatic angiography reveals high vascularity of the tumour.

5- Chest X ray, CT scan & bone scan to detect distant metastasis.

Differential diagnosis:

From other space-occupying lesion of the liver, especially tender ones.

Treatment:

I- Operable cases:

-Few cases are operable and can be resected.

-Segmentectomy, lobectomy or hemihepatectomy may be indicated.

-Hepatic transplantation is tried but the results are inferior to the standard.

II- Inoperable cases.

1- Systemic chemotherapy, poor response.

2- Arterial chemo-embolization: Intra-arterial injection of chemotherapeutics combined with gel foam embolization through angiography.

3- Radiofrequency ablation of the tumour

4- Intra-lesional injection of ethanol: alcohol causes tumour necrosis.

5- - Radiotherapy palliative to reduce pain.

Prognosis:

It is very poor.

Sarcomas of the liver

- They are very rare
- They grow rapidly. They may rupture causing serious intra-peritoneal haemorrhage.
- The best management is early surgical resection.

Secondary tumours

- They are 20 times more common than primary ones.
- They reach the liver through.

1- Portal vein: e.g. stomach intestine, pancreas, spleen, etc.

2- Hepatic artery: e.g. cancer lung, breast kidney.

3- Lymphatics: e.g. cancer colon cancer breast .

4- Direct route: e.g. carcinoma of the gall bladder.

Pathology:

Metastases usually from multiple, pale white nodules umbilicated due to central necrosis.

Clinical picture:

In addition to the manifestations of the primary tumour, there may be .athenia, anorexia and anaemia .The patient may complain of Rt. hypochondrial pain, progressive jaundice & ascites.

Investigations:

1- Liver function tests: usually reveal high S.bilirubin and S.alkaline phosphatase.

2- Abd. U/S, CT scan or MRI.

Treatment:

I- Surgery (metastatectomy).

Prerequisites

1- Completely resectable primary colorectal carcinoma with no apparent residuals.

2- No extrahepatic metastases.

3- Single liver metastasis or multiple but confined to one segment or surgical lobe

Surgical treatment includes wedge resection, hepatic segmentectomy, lobectomy, or hemihepatectomy (see below).

II- Interventional treatment:

All the methods mentioned in treatment of the primary tumours

Cysts of the Liver

I- Congenital:

1- Fibrocystic disease 2- Dermoid cyst

II- Developmental:

Retention cyst

II- Traumatic:

encysted haematoma (blood cyst)

IV- Infection:

1- Hydatid cyst (the commonest) 2- Abscesses (amoebic or pyogenic)

V- Neoplastic:

A-Benign: 1- Cystadenoma

2-Heamangioma & lymphangioma (lymphatic cyst)

B- Malignant: Cystic degeneration of malignant tumour

Hepatic resection

Resection of one or more hepatic lobes is now possible without interference with the function, vascularity or biliary drainage of the remaining other lobes .

Indications:-

1- Massive liver trauma to a segment of the liver .

2- Huge cysts e.g hydatid , amoebic abscess .

3-Benign tumours huge adenoma or hemangioma .

4- Malignant tumours :-

1] Primary : hepatoma , cholangiocarcinoma

2] Secondary: - local spread from gall bladder or gastric cancer

- Blood localized metastases from resectable colorectal cancer

Types :-

(1) Wedge resection: in small benign tumour or localized metastasis

(2) Left lateral segmentectomy: removal of segment II , III (the left morphological lobe)

(3) Left hepatectomy (left hepatic lobectomy) : removal of the liver lying to the left of the main fissure , and middle hepatic vein

(4) Right hepatectomy (right hepatic lobectomy) : removal of the liver lying to the right of the main fissure and middle hepatic vein .

(5) Extended right hepatectomy (Trisegmentectomy) : excision of the right surgical lobe + segment IV , the commonest indication is carcinoma of the gall bladder .

Preoperative preparation :-

- Improve the nutritional status of the patient with high CHO & protein diet.
- Correct any hypoalbuminaemia , electrolyte disturbance or coagulopathy .
- Correct anaemia or thrombocytopenia if present .

Complications

[I] Intra-operative :-

1) Shock usually hypovolaemic

2) Haemorrhage : serious bleeding may occur from hepatic artery or portal vein branches or more seriously inferior vena cava .

3) Injury to important structures e.g duodenum, right adrenal, stomach or biliary tree of the remaining segments.

4) Difficulty : especially in cirrhotic liver or presence of adhesions surrounding the porta hepatis

[II] Post-operative :-

1) Biliary leakage causing biliary fistula or bilomas

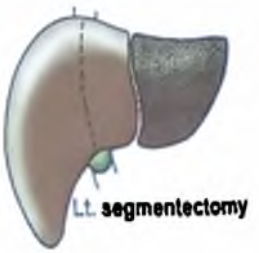
2) Hypoglycaemia: give glucose 10 %

3) Hypoalbuminaemia: give human albumin

4) Coagulopathy such as hypoprothrombinaemia: give fresh frozen plasma

5) Infection: give antibiotic

6) Liver cell failure (see portal hypertention)



BILIARY TRACT

Anatomy of the gall bladder

It is a pear shaped sac 50 cc in capacity, lies in its fossa on the undersurface of the liver. It is divided into:

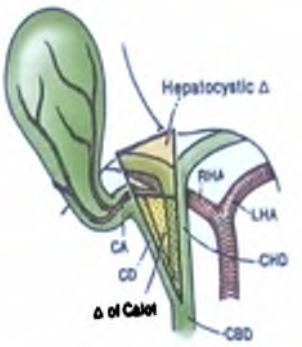
1- Fundus: the part projecting beyond the lower border of the liver.

2- Body: the capacious part

3-The neck: the narrow terminal part that is continuous with the cystic duct and has infundibulum called *Hartmann's pouch.*

Surface anatomy of the gall bladder:

The fundus of the gall bladder lies in the angle between the tips of the Rt. ninth costal cartilage with the Rt. vertical plane.



Anatomy of the gall bladder

The cystic duct: S shaped duct 2-4 cm in length, connecting the gall bladder to the common hepatic duct (CHD) .The triangle formed by the cystic duct, cystic artery & common hepatic duct is called *Callot's triangle*, which has a special importance during the operation of cholecystectomy for identification of these structures.

Blood supply:

Arterial: cystic artery from the right hepatic artery.

Venous: drains directly into the liver or Rt. branch of the portal vein.

Lymphatic drainage: passes first to the cystic lymph node which lies on the junction of the cystic & common hepatic duct , then to hepatic L.N.s at porta hepatis → coeliac L.N.s .

Nerve supply

Autonomic: both sympathetic & parasympathetic from the coeliac nerve plexus.

Somatic: additional fibres from the right phrenic nerve.

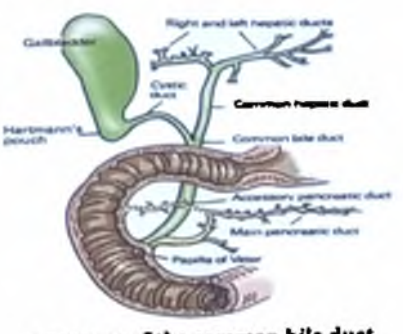
Anatomy of the common bile duct

It is 8-cm long tube formed by the junction of the cystic & CHD. It is divided into 4 parts:

1- ***Supraduodenal part***: passes in the free (Rt.) border of the lesser omentum right to the common hepatic artery and infront of the portal vein. .

2- ***Retroduodenal part***: behind the first part of the duodenum.

3- ***Infraduodenal part***: Lies in a groove in the posterior surface of the head of pancreas.



Anatomy of the common bile duct

4-Intraduodenal part: In about 85% of persons, the common bile duct joins the main pancreatic duct to form (a common channel) 1 cm long which has an intraduodenal dilatation called hepatopancreatic ampulla or *Ampulla of Vater*

Applied Anatomy

1- Stone impacted in Hartmann's pouch may obstruct both the cystic & common bile duct.

2- Also stone impacted in the distal end of CBD obstruct both CBD & pancreatic duct predisposing for pancreatitis

3- The cystic duct's mucosa is folded in a manner to form a spiral valve called spiral valve of Hiester that may hinder the flow of bile after cholecystojojunostomy.

4- The bl. vessels of the common bile duct run longitudinally in the wall especially at 3 & 9 O'clock, so the duct should be explored by a longitudinal incision at 12 O'clock so as not to damage these vessels and to avoid stricture.

I- Congenital anomalies

I- Anomalies of the gall bladder.

A- Anomalies of number.

1- Absent gall bladder.

2- Double gall bladder, the two bladders either open separately or into one cystic duct.

B- Anomalies of septum formation.

1- Phrygian cap: a septum at the fundus of the gall bladder making it similar to hats worn by people of phrygia.

2- Septate gall bladder: one or more septa divide the gall bladder into compartments.

C- Divertucula of the gall bladder.

D- Floating (mobile) gall bladder:

The gall bladder is completely covered with peritoneum and has a mesentry making it hypermobile and liable to torsion.

Double gall bladder

Phrygian cap

Septate gall bladder

Insertion into the Rt.HD

Low insertion into CBD

Diverticulum in GB

II- Anomalies of cystic duct .

A- Anomolies of number.

1- Absent cyst duct (sessile gall bladder)

2- Double cystic duct.

B- Anomalies of insertion.

1- Insertion into the right hepatic duct.

Cholecysto-hepatic duct

Seg. IV drains into cystic duct

2- Low insertion into the common bile duct:

III- Anomalies of the cystic artery

A- Abnormal origin of the cystic artery.

It may arise from the left or common hepatic artery, gastroduodenal artery or even the sup. mesentric artery .

Monyhan's hump

Accessory cystic artery

B- Accessory cystic artery.

It arises from the common or the right hepatic artery and cause bleeding during cholecystectomy.

C- Monyhan's hump or caterpillar turn:

The right hepatic artery may have a tortous course in front of the cystic duct making it liable to injury during cholecystectomy.

IV- Anomalies of the bile ducts .

A- Cholecystohepatic duct: An accessory duct that passes directly from the liver into the gall bladder. It is not uncommon and may causes postoperative biliary leakage that usually stops spontaneously

B- Drainage of segment IV directly into the cystic duct. The hepatic segment IV or the right posterior sectorial duct may drain directly into the cystic duct or the neck of gall bladder.

C- Congenital biliary atresia.

Aetiology :

It is an inflammatory process rather than congenital anomalies that destroy the bile ducts. It begins around the time of birth and continues.

Type I

Pathology:

Types .

1- Type I: Atresia restricted to the common bile duct.

2- Type II: Atresia extends to the common hepatic duct.

3- Type III: Atresia extends to the Rt. & Lt. hepatic ducts.

Type II

Complications and causes of death :

1- Progressive obstructive jaundice.

2- Biliary cirrhosis. & Portal hypertension.

3- Associated congenital anomalies e.g. cardiac lesions, polysplenia, situs inversus, absent vena cava .

Type III

Incidence:

It occurs in one per 14000 live births. It occurs equally in both sexes.

Clinical picture :

1- Jaundice : 1/3 of cases are jaundiced at birth but all cases are jaundiced by the end of the 1st week.

2- Steatorrhea; Early, the meconium is little bile stained but later the stool will be pale and the urine is dark. Prolonged steatorrhea will lead to osteomalacia (biliary rickets) .

3- Pruritis and finger clubbing.

5- Picture of complications.

Investigations:

1- Estimation of liver function: Raised S. direct bilirubin, S. alkaline phosphatase and liver enzymes.

2- HIDA scan or better MRCP: It shows the anomaly, the site of obstruction and the type of atresia.

Differential diagnosis:

From other causes of neonatal jaundice.

Treatment :

Type I :

It may be corrected by Roux-en-Y jejunal anastomosis with the common hepatic duct. It succeeds in 75% of cases to establish biliary flow.

Type II and III:

1- Resection of the left lobe of the liver and the dilated ducts in the cut surface are anastomosed to a Roux en-Y lop of jejunum (hepatico jejunostomy).

2- *Kasai operation* (porto-jejunostomy) : excision of the whole extra-hepatic bile duct tissue then a Roux en Y loop of jejunum is anastomosed to the liver capsule at porta hepatis .

3- Liver transplantation should be considered in the failed cases.

Prognosis:

- Even in successful surgery, progressive fibrosis continues and impairs the prognosis in long term results. Most cases die around the age of 3 years

D- Choledochal cyst.

Aetiology:

Congenital weakness of a part or the whole of the bile duct leading to dilatation and stasis .

Pathology:

Types .

Type I:

Ia: cystic dilatation of the CBD & CHD

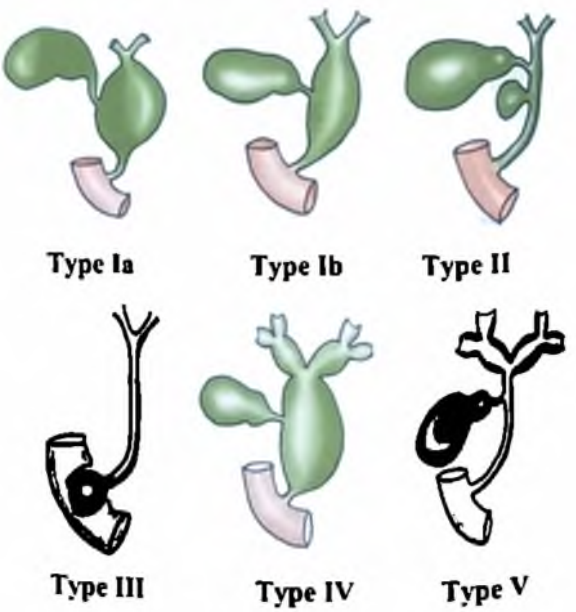
Ib: Fusiform dilatation of the CBD & CHD .

Type II: Diverticulum from the CBD.

Type III: Dilatation of the infra-duodenal portion of CBD (cholodochocele).

Type IV: Dilatation of the CBD and CHD and the two hepatic ducts.

Type V: Dilatation of the hepatic ducts and the intrahepatic ducts (Caroli disease).



Complications:

1- Obstructive jaundice with all its complications.

2- Recurrent cholangitis .

3- Acute pancreatitis due to associated long common channel.

4- Malignant transformations: carcinoma of the bile duct with a bad prognosis .

5- Rupture : leading to acute abdomen .

Clinical picture :

The patient may present with.

1- Jaundice: obstructive jaundice.

2- Swelling: upper abdominal swelling in the epigastrium and right hypochondrium .

3- Complications (mentioned above).

Investigations:

1- Abdominal U/S

2- CT & MRI.

3- MRCP is of choice.

Treatment:

-The treatment of choice is excision of the cyst because of its malignant potential then reconstruction of Roux-en-Y choledocho-jejunostomy or heptico-jejunostomy.

-Other methods as cysto-enterostomy was found to be ineffective, associated with repeated cholangitis and it does not eliminate the risk of malignancy.

II-Trauma

Bile duct injury (see surgical notes for postgraduates)

III- Inflammatory conditions

Acute cholecystitis

Aetiology:

Predisposing factors:

Obstruction of the cystic duct: is usually the initiating factor in most cases (about 90%). Obstruction leads to more concentration of bile inside the gall bladder → chemical irritation of the mucosa →2ry bacterial invasion.

Causative organisms:

-E- coli (commonest), Klebsilla, strept. faecalis & Bacteroids .

-Clostridia. welchii → *emphysematous cholecystitis* .

-Salmonella. typhi → *typhoid cholecystitis* .

Routes of infection:

1- *Direct spread.* along the bile ducts .

2- *Haematogenous* from distant focus e.g. typhoid cholecystitis.

Pathology:

I-Obstructive cholecystitis (85-95%): Has the following stages:

1- *Catarrhal inflammation*: localized to the mucous membrane with congestion of the other coats. The gallbladder is distended with mucous & bile (*mucocele*).

2- *Suppurative inflammation*: 2ry bacterial invasion usually occurs & the gallbladder becomes a bag of pus (*empyema*). Omentum & other viscera soon become adherent to it.

3- *Gangrenous stage*: if the obstruction is not relieved (by disimpaction) and the inflammation is severe, septic thrombosis of the vessels occurs with gangrene and perforation. Perforation occurs either at the fundus or at the neck.

II- Non-obstructive (5-15%):

It passes through the same stages with the following differences:

1- Mucocele & pyocele do not occur.

2- The clinical picture is milder & the progress of events is slower.

3- The gangrene & perforation are far less common.

Complications:

1- Chronicity :

The commonest complication. Acute inflammation subsides & the gall bladder becomes the seat of chronic inflammation and stones formation.

2- Perforation: rare (why?):

a) The gall bladder is capacious and distensible.

b) The blood supply is rich.

c) The thick fibrosed wall of the bladder from previous ch. inflammation will resist perforation

It results in either:

A-Local abscess: the commoner, as adhesions with omentum & other viscera shut off the infection from the rest of the peritoneal cavity. It may:

- Burst into hollow viscus (e.g. colon or duodenum) causing internal biliary fistula.
- Burst to the exterior (through the skin causing ext. biliary fistula.

B-Generalized peritonitis.

3-Stone formation:

4-Ascending cholangitis & liver abscesses.

Incidence:

Common in **F**atty, **F**latulent, **F**ertile **F**emale about **F**orty years (5 **F**s).

Clinical picture: -

History: the patient usually gives history of attacks of biliary colic or cholecystitis.

Symptoms:

1-General constitutional symptoms fever, anorexia, malaise.

2-Pain

-Type : colicky -Intensity : severe

-Site :- Rt. hypochondrium -Radiation : to the Rt. shoulder & Rt. scapula

-increased by : movement, coughing & deep breathing

3- Nausea & vomiting.

Signs:

I-General

- Fever, tachycardia
- Tinge of jaundice may be present due to :

1-stone impacted in Hartmann's pouch compressing the C.B.D or *Mirizzi syndr*ome

2-Double impaction i.e. another stone in the C.B.D.

3-Associated cholangitis.

Mirizzi syndrome

II- Abdominal:

1- Tenderness & guarding in the Rt. hypochondrium.

2-*Boas sign*: hyperesthesia between the 9th and 11th rib on the right side posteriorly.

3-The gall bladder may be palpable in case of mucocele or pyocele as:

- A tender tense cystic pyriform swelling in the right hypochondrium.
- Moves up and down with respiration.
- Smooth surface and rounded border.
- Not ballotable.
- Dull on percussion and its dullness is continuous with hepatic dullness.

Investigations:

1-Leucocytic count: usually moderate leucocytosis.

2-Ultrasonography: very helpful.

3-I.V. cholecystography (not reliable) or better Radio-nucleotide scan (HIDA) scan. 99 Tcm labeled derivatives of imino diacetic acid are excreted in bile within 15-30 min after I.V Inj.

.If the gall bladder is visualized, diagnosis of acute cholecystitis should be revised.

Differential Diagnosis:

From other causes of acute abdomen specially:

-Acute pyelitis -Liver abscess -Subhepatic appendicitis

Treatment:

I-Delayed surgical treatment = conservative treatment.

Conservative measures are undertaken at first .If the patient improves within 48 hours, medical measures are continued and cholecystectomy is performed 3-4 weeks later.

Conservative measures are:

1- Hospitalization. *2- Bed rest in semisitting position.*

3- N.P.O + I.V fluids. *4- Nasogastric suction.*

5- Antibiotics. *6- Antispasmodic.*

7-Hot applications. *8-Observation.*

If the general & local observations worsen, immediate surgical intervention must be performed.

II- Early surgical treatment:

Indications

1- *Failure of conservative measures* i.e. the patient deteriorates within 48 hours.

a- Spreading peritonitis (perforation).

b- Increasing jaundice.

2- *Doubtful diagnosis.*

Operation:

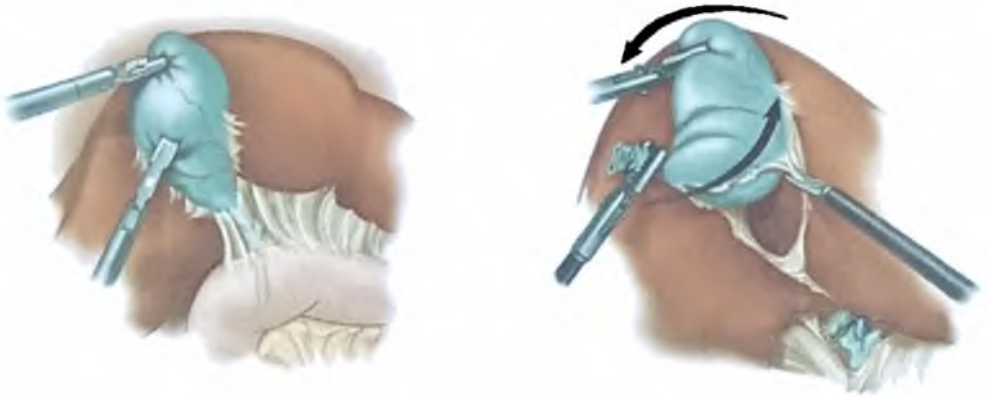
The operation done is cholecystectomy if possible or cholecystostomy.

Indications of cholecystostomy:

1- Poor general condition (under local anaesthesia, percutaneous U/S guided)

2- Cases with marked oedema & adhesions (disturbed anatomy) especially if jaundiced.

Laparoscopic cholecystectomy in acute cholecystitis was tried with great success.



Imaging studies of the biliary system:

1- Plain X ray may show the stone in a low percent of cases.

2- Ultrasound: of great help to show GB. stones, CBD. dilatation & Intrahepatic biliary radicles dilatation (IHBR), mass in the pancreatic head and sometimes stones in CBD.

3-Oral cholecystogram:

The patient is given 10 telepaque tablets at night and radiographed in the morning. Only the GB. appears opacified if it concentrated the dye. Then the patient is given a fatty meal to evacuate the GB. and radiographed after 1-2 h. to show the contractility of the GB.

4-intravenous cholangiogram

20 cc of biligraphin is injected IV. and a X ray is taken 10 minutes later. Normally, the gall bladder and the biliary tree can be visualized.

5- Radio-nucleotide scan (HIDA) scan. 99Tcm labeled derivatives of imino diacetic acid are excreted in bile within 15-30 min after I.V injection

6-Endoscopic Retrograde CholangioPancreatography (ERCP):

Through a duodenoscope, the ampulla of Vater is cumulated and a contrast material is injected under screen to delineate the biliary passages and the pancreatic duct.

7-Precutaneous Transhepatic Cholangiography (PTC.)

Under U/S guide, a needle is introduced percutaneously through the liver to cumulate an intrahepatic duct. Then the contrast material is injected into it to delineate the biliary tree above the obstruction. It is helpful but invasive. It may be combined with **ERCP.**

8- Magnetic Resonance Cholangio-Pancreatography (MRCP):

Now. It is the standard and the best method for imaging of the biliary tree. It does not need contrast material and gives a very good imaging data of the biliary system.

9-Computerized Tomography (CT) Scan: in difficult cases.

10 Intra-operative cholangiogram:

Intra-operative cannulation of the cystic duct, the dye is injected and the patient is X rayed

11-Post operative cholangiogram = T tube cholangiogram: see later

NB. The oral and IV cholangiography are almost obsolete.

Chronic cholecystitis

Definition

It is a clinical term applied to all chronic pathological conditions of the G.B., which include:

1- Chronic calcular cholecystitis.

2- Chronic non-calcular cholecystitis (bacterial infection).

3- Biliary dyskinesia (gallbladder stasis).

4- Cholecystoses (cholesterolosis + cholecystitis glandularis proliferans).

Aetiology & pathology:

Each of the four types has its own causes & pathology:

1-Chronic calcular cholecystitis (gall stones) 95%

Predisposing factors for stone formation:

① *Ch. Inflammation of the gall bladder* provides:

- Good nucleus for stone formation as debris & bacteria colonies.
- Good soil (rough surface): ulcerated mucosa.
- Good climate & manure (suitable media): altered pH & excess albumin.
- Enough time (stasis due to ↓ contractility or obstruction).

② *Disturbed cholesterol metabolism.*

③ *Stasis*: due to spasm, fibrosis or obstruction.

④ Increased *rate of haemolysis* leads to↑↑ concentration of bile pigment → pigment stone.

Types of gallstones:

Items	Cholesterol stone	Mixed stone	Pigment stones	Ca carbonate stone
Predisposing factor	Disturbed cholesterol metabolism.	Ch. inflammation	Haemolytic conditions	Stasis
Incidence	10%	80%	8%	2%
Number	Usually single	Multiple (in groups)	Multiple	Emulsion
Size	Usually large	Variable	Small	Lime
Shape	Oval	Hexagonal, faceted	Irregular	water
Surface	Mammillated	Smooth faceted	Smooth	bile
Colour	whitish	Brown to gray	Dark green or black	White
Consistency	Hard	Hared	Soft	Tooth paste
Cut section	Radiated	Concentric laminations	Amorphous	
- X ray	radio translucent	Usually radiopaque (Signet ring app)	50% are radio-opaque	Radio opaque

N.B. Cholesterol & pigment stones are considered metabolic stones while mixed stone & Ca CO_3 are called secondary stones.

2-Chronic non-calcular cholecystitis:

-Bacterial infection caused by B. Coli, Strept. Klebsiella, S. typhi & Cl. Welchii. Usually reaches the gall bladder through the blood stream from septic focus.

- The gall bladder harbors no calculi but shows criteria of ch. inflammation:

a- Loss of normal serosal luster with adhesions to the surroundings e.g. omentum & colon.

b - Enlarged cystic LN. .

c -Thickened wall with contracted size.

d -Cobweb appearance of the gall bladder bed.

e- Loss of normal mucosal folds .

3-Biliary dyskinesia (stasis gall bladder): There are two varieties

a- Atonic type: in which the gall bladder is lax and not capable of contraction

b- Hypertonic type: in which there is achalasia of sphincter of Oddi leading to, marked dilatation of the gall bladder. Cholecystectomy always aggravates the condition.

4-Cholecystosis: It includes two varieties

A-- Cholesrolosis =Strawberry gall bladder:

Due to error in cholesterol metabolism leading to deposition of the yellowish cholesterol esters on reddened mucosa giving it the strawberry appearance.

B-Cholecystitis glandularis proliferans:

It includes variety of pathological conditions (polyposis, adenomyosis & intramural diverticulosis & calculi) It may be complicated with abscess &fistula.

Complications:

1. Complications of stones:

A- Gall bladder: -Acute & chronic cholecystitis.
-Mucocele, empyema, gangrene, perforation, fistulae.
-Carcinoma.

B- Biliary passage: -Biliary colic & obstructive jaundice.
-Cholangitis, liver abscess.
-Pancreatitis, Mirrizi syndrome (see later).

C- Intestines: -Gall stone ileus.

2- Recurrent acute attacks.

3- Toxic symptoms: toxaemia, headache, malaise, and bony aches.

Incidence:-

It is a common disease especially in Fatty, Filthy, Fertile, Female above the age of Forty (5Fs)

Clinical picture:

Symptoms

1-Fatty dyspepsia: distension, flatulence & heart burn after meals especially fatty ones.

2-Pain

Type: dull aching	Intensity : variable
Site : Rt. hypochondrium	Radiation : Rt. shoulder & back
Increased by : fatty meals	Associated with : nausea & vomiting

3-Symptoms of complications:

Toxic symptoms jaundice … etc.

Signs

1-Mild jaundice may be detected.

2-Tenderness over the tip of the Rt. ninth costal cartilage.

3-Murphy's sign: when the patient is asked is have a deep breath whilst gentle pressure is applied to the gall bladder area, the patient will catch breath before the end of inspiration

4- Boas sign: may be positive.

5-Palpable gallbladder: in case of mucocele.

Associated diseases :

1- *Wilkie's triad*: ch. cholecystitis is often associated with ch. peptic ulcer & ch. appendicitis

2- *Saint's triad*: - Rarely gallstones are associated with hiatus hernia and diverticulosis coli.

Investigations:

1- Abd. Ultrasound: the investigation of choice (see before).

2- Plain X ray: - only about 15% of cases show radio opaque gall stones (D.D of radiopaque shadow in the Rt. hypochondrium.).

3- Oral cholecystogram: If the GB. concentrates the dye, radiotranslucent stones & polyps appear as filling defects (*Honey coomb appearance*). Another film is taken after fatty meal to test the GB. contractility

4- IV. Cholecystogram: The dye is injected IV. and the GB. is radiographed after 10 minutes. It shows in addition the intrahepatic biliary radicles

5- Cholecystokinin cholecystogram: is an oral cholecystogram with injection of Cholecystokinin. In biliary dyskinesia, the gall bladder assumes *a football configuration*.

6- Duodenal aspiration: diluted bile (failure of G.B. to concentrate bile).

Differential diagnosis:

1- Ch. peptic ulcer 2-Ch. appendicitis 3- Hiatus hernia

4-Ch. pancreatitis 5- Spastic colon

Treatment: -

I- Conservative ttt

A-Medications:

1- *Diet* : restriction of fat 2- Antibiotics

3-Cholagogues : (i.e. evacuate the G.B.) e.g. olive oil or Mg sulphate

4-Choleretics (i.e. increase bile secretion) e.g. bile salts

5- *Antispasmodics* 6- *Analgesics*

B- Oral dissolution therapy

Chenodeoxycholic acid &Ursodeoxycholic acid dissolve cholesterol stones.

Prerequisites:

1- The stones should be pure cholesterol stone & non-calcified (radiotranslucent).

2- Stones should be less than 4 stones and each less than 1.5 cm in diameter.

3- The cystic duct should be patent with functioning G.B.

Success rate is about 50% after 2 years but recurrence rate is 50% within 5 years

II- Lithotripsy + dissolution

Fragmentation of stones using Extracorporeal Shock waves Lithotripsy (ESWL) to smaller fragments amenable to dissolution therapy.

III- Percutaneous cholecystolithotomy:

For patients who refused cholecystectomy, percutaneous removal of the stones is advised

IV - Surgical ttt = cholecystectomy

Indications

1- Chronic calcular cholecystitis

2- Ch. Non-calcular cholecystitis with failed medical ttt for at least 6 months or with repeated acute episodes.

Types

1- Open cholecystectomy:

Incisions

1- Kocher's subcostal incision in fatty patients

2- Rt. upper paramedian incision in thin patients

3- Minilaparotomy cholecystectomy :small (2 inches) transverse incision over the rectus

2-Laparoscopic cholecystectomy

Through 4 small incisions (stabs). The 1st is umbilical for the camera, the 2nd in the epigastrium for the operator and two small lateral right incisions for the assistants .Finally after dissection of gall bladder, it is delivered through the epigastric incision

Indications of exploration of the C.B.D. (choledochotomy) during cholecystectomy

A- Preoperative indications :

1- History of Jaundice or Charcot's triad.

2- Ultrasonographic evidence of CBD. stones or dilatation > 1 cm .

3- High S. alkaline phosphatase especially > 30 King Armstrong.units.

B- Intra-operative indications :

1- Palpable stone in CBD (absolute indication).

2- Abnormally dilated. CBD or if aspiration revealed biliary mud.

3- Operative cholangiogram revealed stone or stricture C.B.D.

Complications of cholecystectomy

I- Intra-operative complications:

1- Shock

2- Hemorrhage: commonly due to injury of the cystic artery, rarely due to injury of major bl. vessels e.g. portal vein or Rt. hepatic artery especially in Moynihan's hump anomaly

3-Injury of important structures e.g. CBD, duodenum

4- Difficult dissection of the GB. due to adhesions (liver cirrhosis) → Partial cholecystectomy

II- Postoperative complications:

1 Obstructive jaundice: due to closed injury or accidental ligation of CBD.

2- Biliary fistula: due to open injury of CBD or slipped ligature of the cystic duct.

3- Portal vein thrombosis: due to closed injury of the portal vein

4- Postcholecystectomy syndrome

Definition: persistence of dyspeptic symptoms after cholecystectomy

Aetiology:

a- Wrong diagnosis: Dyspepsia for other causes peptic ulcer, hiatus hernia, spastic colon or biliary dyskinesia

b- Wrong technique: injury to C.B.D or missed stone.

c- Wrong patient: neurotic patients who always complain.

5-General complications of abd operations e.g. pulmonary complications, paralytic ileus, burst abdomen and post operative infection

PANCREAS

Embryology

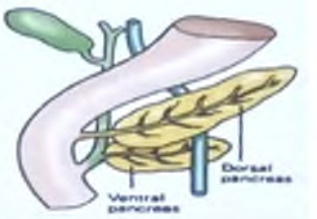
-The pancreas develops from two processes (buds):

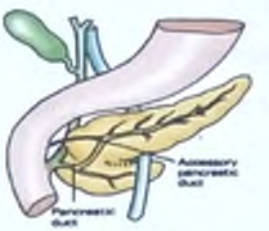
1-Ventral pancreatic bud from hepatic bud (that forms the CBD).

2-Dorsal pancreatic bud from the 2nd part of the duodenum.

-The ventral pancreatic bud migrates dorsally to fuses with the dorsal one and forms the uncinate process of the pancreas.

-The ventral pancreatic duct communicates with the dorsal one by small communicating duct to form the main pancreatic duct & the proximal part of the dorsal duct obliterates & disappears or persists as the accessory pancreatic duct.





Development of the pancreas

Anatomy

Pancreas is a thin elliptical gland in the retroperitoneum of the upper abdomen (epigastrium & Lt. hypochondrium).

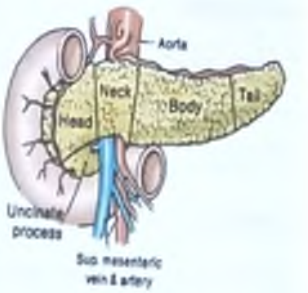
Anatomical parts:

1- Head: - lies in the concavity of the duodenum, and has a hook like process called (*Uncinate process*).

2- Neck: is a narrow part lies behind the 1st part of duodenum.

3- Body: is prismatic in shape and lies posterior to lesser sac.

4- Tail: passes through the lienorenal lig. to splenic hilum .



Anatomical parts

The duct system of the pancreas:

The pancreas drains into the duodenum with two ducts:

1-Main pancreatic duct (wirsung).

It is formed by the fusion of most of the dorsal duct & ventral duct. It usually (in 85% of populations) fuses at its distal end with the C.B.D to form the ampulla of Vater, which opens in the second part of the duodenum through the major duodenal papilla.

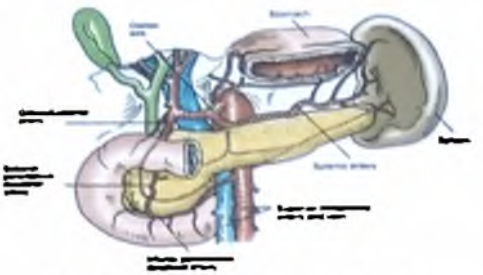
2- Accessory pancreatic duct. *(Duct of Santorini)*:

It opens into the duodenum through the minor duodenal papilla

The duct system of the

Blood supply

The pancreas has its blood supply from branches of the splenic artery and superior & inferior pancreatico duodenal arteries. Venous drainage is parallel to arteries.



Blood supply of the pancreas

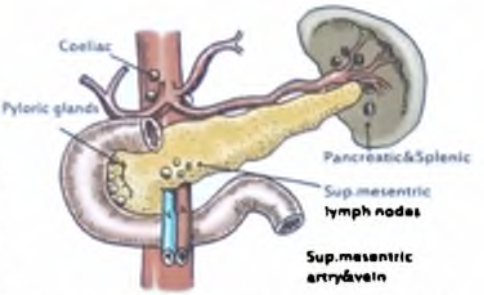
Nerve supply:

Parasympathetic (secretory), by the vagus

Sympathetic, splanchnic nerves

Lymphatic drainage

Lymphatics of the pancreas drain into pyloric L.N (between the head of pancreas and duodenum), pancreatico-splenic L.N.s, which end in celiac and sup mesenteric groups of pre-aortic L.N/S.



Lymphatic drainage of the pancreas

I- Congenital Anomalies

1-Annular pancreas

It is due to abnormal fusion of ventral and dorsal pancreas to form a collar of tissue surrounding the second part of the duodenum and may compress it

Clinical picture: sometimes, it is symptomless but it may be complicated with duodenal obstruction or peptic ulceration.

Investigations:

Plain X ray in erect position →Duodenal obstruction → double bubble sign.

Treatment: -

Relieve the obstruction by Duodenojejunostomy or Duodenoduodenostomy.

3-Cystic fibrosis of pancreas (see meconium ileus).

4-Ectopic pancreatic tissue.

5-Pancreatic divisum: persistence of the embryological condition duo to failure of fusion of the ventral & dorsal pancreatic ducts.

II – inflammation

1- Acute pancreatitis

Definition:

It is acute condition due to inflammatory disease of pancreas.

Aetiology:

Autodigestion of the pancreas following activation of its proteolytic enzymes e.g. trypsinogen.

Causes (BAD TIME)

1-Biliairy pancreatitis: Obstruction of the ampulla of Vater causes regurgitation of bile into the pancreatic duct (common channel) with activation of pancreatic enzymes starting autodigestion. This theory explains about 50% of cases.

Common channel theory

2-Alcoholism: 25% of cases are alcoholic may be due to hyper-secretion or spasm of sphincter of Oddi

3-Drugs: many drugs were incriminated

4-Trauma: either blunt trauma to epigastrium or, sharp trauma e.g. stab or iatrogenic surgery.

5-Infection: either blood borne or complicate mumps (viral pancreatitis).

6-Metabolic disorders: DM, hypercalcaemia, porphyria

7-ERCP

Pathology:

Types:

1-Acute oedematous pancreatitis: It is mild form with oedema and congestion of the pancreas.

2-Acute haemorrhagic pancreas: It is more severe form with autodigestion and escape of pancreatic enzymes to the peritoneum leading to:

a) Erosion of bl. vessels with haemorrhagic exudate.

b) Digestion of fat by lipase enz. liberating the free fatty acids which react with Ca^{++} in the exudate $\rightarrow Ca^{++}$ soap which appear as dull whitish yellow waxy patches in the mesentry.

1- Acute narcotizing pancreatitis :-

It is the most severe form with partial or complete necrosis (bad prognosis).

Complications: -

1-Hypovolaemic shock: It is due to marked loss of fluid in the peritoneum and retroperitoneum.

2-Hypoxia: - Mild hypoxia with or without cyanosis may occur due to respiratory insufficiency.

3-Hypocalcaemia: It may be due to binding of Ca^{++} in soap or due to hypoalbuminaemia.

4- Sepsis: Infection of necrotic pancreatic tissue may cause pancreatic abscess.

5- Stricture of the transverse colon: It may occur in the segment, which overlies the pancreas.

6- PSeudocyst formation: accumulation of fluid (exudate) in the lesser sac with occlusion of foramen of Winslow.

Incidence:

Common in middle age. Male = Female common in alcoholics & diabetic

Clinical picture: -

Symptoms: -

1. Pain: severe epigastric pain radiating to the back.
2. Vomiting: reflex vomiting & due to gastric irritation.
3. Collapse: may be with cyanosis.

Signs:

General:

1- Signs of shock (hypotension, tachycardia).

2- Mild cyanosis. 3- Faint jaundice.

Local:

1. Tenderness and rigidity maximum over epigastrium.
2. Shifting dullness.
3. Ecchymotic blue discolouration in the flanks (Grey Turner sign) ,or around the umbilicus (*Cullen 's sign*) may appear after 2-3 days.It is due to diffusion of pancreatic enzymes from the retropertoneum to the flanks or through the falciform ligament of the liver .

Investigations:

1- Serum amylase (N = 90 – 150 Somogyi units / 100 ml).

- It rises to a very high level after an hour then falls to normal level within 3-5 days.
- To be diagnostic, it should be at least four times its normal value
- The degree of enzyme elevation does not correlate with disease severity

Pancreatitis without hyperamylasaemia	Hyperamylasaemia without pancreatitis
1. After 24 hours	1. Intestinal obstruction
2. Hyperlipidaemia	2. Perforated peptic ulcer
3. Acute total necrotizing pancreatitis	3. Acute cholecystitis
4. Chronic. pancreatitis	4. Mesenteric vascular occlusion
	5. Sialadenitis (macroamylasaemia)

3. Urinary amylase: (N- 30-50 units/hour).

It rises to 100 – 500 units after –12-24 h. and falls more gradually.

4. Serum lipase (N=0-1.5 cherry – Crandall units/100 ml): more specific.
5. Serum Ca^{++}: is usually low mostly from the nonionized part, (tetany is rare).
6. US & CT. scan: show oedematous pancreas, associated biliary disorder & peritoneal exudate

Differential Diagnosis:

From other causes of acute abdomen especially perforated peptic ulcer and acute cholecystitis.

Treatment: -

I- Conservative treatment: is the mainstay measure:

1. Aggressive fluids resuscitation with close monitoring of.heart rate, blood pressure, urine
2. Analgesic: pethedine not morphia
3. Antibiotics: to guard against secondary infection (controversial).
4. Antisecretory drugs e.g. Somatostatin in full dose.
5- Enteral feeding should be started as early as possible e.g. once vomiting stops.
6. Correction of electrolytes imbalance:e.g. Ca^{++} gluconates infusion.

II- ERCP and stone extraction for proven bile duct stones causing obstruction and pancreatitis

III- Surgical treatment:

-It was thought that once diagnosis of acute pancreatitis is made surgery is contraindicated, however this belief has recently changed and surgery has definite indications: **(4Ds)**

1- **D**oubtful diagnosis

2- **D**ecompression of the biliary tree if a stone is impacted at the ampulla (if ERCP failed)

3- **D**ebridment of necrotic pancreatic tissue in necrotizing type (pancreatic necrosectmy)

4- **D**rainage of pancreatic abscess or pseudocyst

-If exploration was done in doubtful cases, the diagnosis is made by finding hemorrhagic exudate, soap patches, and oedematous pancreas.

- Drain the lesser sac to prevent pseudocyst formation.
- Closure of the abdomen should be done with non-absorbable sutures.

Prognosis:

- Ranson had stated criteria for estimating the severity of the disease such as the age of the patient, leucocytic count, bl. glucose level, S. calcium and others. These criteria have been modified by Glascow university into Glascow criteria..
- Generally, the prognosis is good if fewer than three Ranson's criteria are present while, patient with more than six criteria rarely survive.
- The overall mortality rate is about 10 %.

2- Chronic pancreatitis

Definition:

Chronic inflammatory disease characterized by irreversible progressive destruction of pancreatic tissue

Aetiology:

1- Many cases are idiopathic.

2- Alcohol consumption is a frequent cause.

3- Pancreatic duct occlusion by stricture due to previous trauma or previous acute pancreatitis.

4- Hereditary pancreatitis: There is a strong evidence of the familial tendency of chronic pancreatitis.

Pathology:

MAC : There are two types

1- Chronic relapsing pancreatitis: Recurrent attack of mild acute oedematous pancreatitis. The organ is enlarged may be with patches of fat necrosis

2- Chronic fibrosing pancreatitis: The pancreas enlarges and becomes hard in consistency due to fibrosis. The ducts become dilated with alternating strictures and ectasia. Stones may be present

MIC :

- The lesion affects the lobules causing ductular metaplasia and atrophy of the acini.
- There is hyperplasia of the duct epithelium with interlobular fibrosis.

Complications:

1- Pancreatic exocrine insufficiency: due to marked damage of pancreatic parenchyma and obstruction of the duct by strictures and/ or stones. It leads to steatorrhea with weight loss.

2- Diabetes mellitus: due to damage of B cells in islets of Langerhans..

3- Obstructive jaundice

4- Insomnia due to pain and disturbance of life and work.

5- Malignant changes: especially in long-standing cases.

Incidence:

- It is more common in middle age (about 40 years). It is more common in males (♂ : ♀ = 4 : 1).

Clinical picture:

History of acute pancreatitis with repeated attacks and hospital admissions may be positive.

Symptoms:

1- Pain:

-*Character*: dull aching -*Intensity*: moderate to severe.

-Site: if it is in the head it is epigastric and if it is in the body it is in left subcostal and the back.

-*Radiation*: to the back in most cases. Radiation to the left shoulder is also common.

-Relief: The patient may feel better with leaning forwards.

*Pain may cause insomnia, disturb work, and cause analgesics abuse.

2- Picture of complications e.g. steatorrhea,

Investigations:

I- Laboratory investigations .

1- Estimation of serum amylase: It is elevated only in the early disease.

2- Tests for exocrine functions.

a) *Secretin and cholecystokinin test*: It is the most sensitive test but difficult.

b) *Faecal fat balance test*: The patient ingests a diet containing 75-100 gm of fat for 5 days. In pancreatic insufficiency faecal excretion of fat may exceed 25%.

3- Blood glucose, S. bilirubin and S. alkaline phosphatase.

II- Imaging studies:

1- Plain X ray: It may show pancreatic stone or calcifications in rare cases.

2- Abdominal U/S., spiral CT.& MRCP: They may show enlarged pancreas and dilated biliary tract in jaundiced patient.

3- Endoscopic retrograde cholangio-pancreatography (ERCP): It may reveal pancreatic stones or strictures of the pancreatic duct alternating with dilatations (Chain of Lakes appearance).

Differential diagnosis:

It should be differentiated from carcinoma of pancreas other causes of obstructive jaundice.

Treatment:

I- Conservative treatment:

1- Stopping alcohol is very beneficial. Smoking should be avoided.

2- Diet low in fat rich in other nutrients is supplied.

3- Morphia is contradicted as it causes spasm of sphincter of Oddi

4- Supplementation with pancreatic enzymes.

5- Diabetes is controlled with insulin.

Puestow procedure

II- Surgical treatment:

Surgery is indicated in

1- Failed conservative measures to bring relief.

2- Cases of duct strictures, stones or obstructive jaundice.

3- Suspicion of malignancy.

Distal pancreati jejunostomy

One of the following procedures.

1- *Longitudinal pancreatico - jejunostomy* (Puestow procedure).

The pancreatic duct is split longitudinally from the tail to the head and anastomosed side to side with Roux en Y loop of jejunum.

2- *Distal pancreatico-jejunostomy (Child's operation).*

- The body and tail of pancreas are removed & anastomosing the head to Roux en Y loop of je

3- Total pancreatectomy or pancreatico-*duodenectomy (Whipple's op.)*

Pancreatic cysts

A- Pseudocyst (80 %):

Definition: accumulation of fluid in the lesser sac with occlusion of foramen of Winslow.

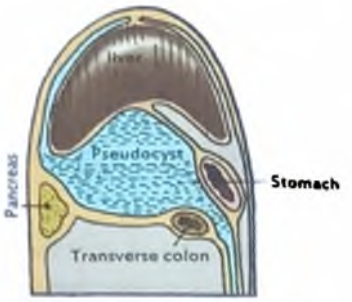
Aetiology: -

1- Acute pancreatitis. 2- Pancreatic trauma.

Pathology: -

1-Acute pseudocyst: follow about 50% of cases of acute pancreatitis. 20 – 40% will resolve spontaneously.

2. Chronic pseudocyst: persist for 2 months after acute attack. They have thick mature walls and liable for complications.



Pancreatic pseudocyst

Complications :

1-Rupture. 2-Infection→ abscess. 3-Hemorrhage. 4-Jaundice.

Clinical picture:

1-History of pancreatitis or pancreatic trauma.

2-Epigastric swelling which is fixed, tense, and cystic with transmitted pulsation from abd. aorta. Pulsation diminishes or disappears in knee elbow position.

Barium meal

Investigations:

1. Barium meal: attenuation of the dye in the lesser curve.
2. U/S, CT, And MRI: make sure diagnosis.

Differential Diagnosis:

1- Mesenteric cyst (Tillaux triad).

2-Aortic aneurysm (knee elbow position).

Treatment:

I- Acute pseudocyst: →conservative.

20 –40 % resolve spontaneously.

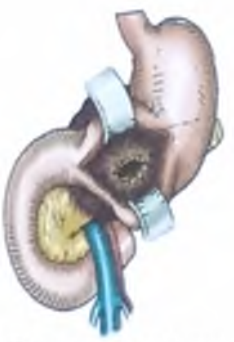
II- Chronic pseudocyst:

1-They will not resolve spontaneously.

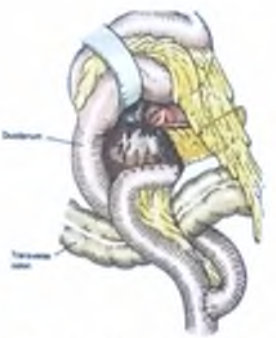
2-They are liable to complications.

3-They have well mature walls that permit internal drainage without risk of leakage.

The operation is cystogastrostomy or cystojejunostomy with long Roux en Y loop



Cystogastrostomy



Cystojejunostomy

B- True cyst (20%):

1. Cystadenoma. 2. Hydatid cyst. 3. Retention cyst. 4. Cystic fibrosis of the pancreas.

IV- Pancreatic tumours

A- Endocrine cell tumours (Islet cell tumours)

1- Beta cell tumour (Insulinoma) The commonest.

. The cells secret insulin → (*Whipple s triad*):

1. Attacks of fainting and may be convulsion during fasting or exercise.
2. The blood glucose level is below (45mg %) during the attack.
3- The symptoms are relieved by ingestion or injection of glucose.

2- Non Beta cell tumours :

They may be gastrinoma (ZE. Syndrome type II), glucagonoma, and vipoma. etc.

B- Exocrine cell tumours

Pancreatic carcinoma

Aetiology:

1- Smoking: there is increased incidence among heavy smokers.

2- Diet rich in protein & fat.

3- Chronic pancreatitis is a premalignant condition.

Pathology:

Site: 1-Cancer head (2/3 of total): 66%

a-Cancer head proper (2/3) .44%

b- Periampullary carcinoma (1/3):22%

i) Duodenal papilla ii) ampulla of Vater iii) duodenal mucosa.

2. Cancer body & tail (1/3 of total).34%

MAC: usually small mass, hard in consistency infiltrating the tissues around.

scirrhous in nature but may be medullary .

MIC: Adenocarcinoma scirrhous or medullary, multicentric in 20% of cases.

Spread: -

-Direct spread: CBD, duodenum, pylorus, IVC.

-Lymphatic spread: LNs between the head and duodenum then to coeliac & sup. mesenteric LNs → thoracic duct →Virchow's gland .

Retrograde spread to LNs at porta hepatis may occur.

-Blood spread: late to the liver.

-Peritoneal implantation in 10% of cases.

Complications and causes of death:

1. Cachexia.
2. Malignant obst. jaundice.
3. Malignant ascites.
4. Duodénal & pyloric obstruction.
5. IVC obstruction.
6. Distant metastasis.

Signs of cancer head

Incidence:

It is commoner in old male (> 60 y).

Clinical picture:

I- Cancer head (jaundice presentation):-

Symptoms:

1. Progressive obstructive jaundice: gradual onset without biliary colic.
2. Pain: may be absent or dull aching in epigastrium referred to back.
3. Pancreatic dysfunction: steatorrhea and indigestion.
4. Anorexia, Asthenia & Anaemia.

Signs:

1- Hepatomegaly: due to obst jaundice. and metastasis .
2- Splenomegaly (in 10%) due to splenic vein thrombosis.
3- Palpable gall bladder: in 60% of cases (Courvoisier law see later).
4- Palpable mass in epigastrium with transmitted pulsation.
5- Ascites: due to malignant implantation &liver seconderies
6- Oedema lower limb due to I.V.C obstruction.
7- Trousseau's sign (migrating superficial thrombophlebitis).
8- Troisier's sign (enlarged supraclavicular L.N (Virchow's gland).

II- Cancer body and tail: (painful presentation):

Symptoms:

1- Severe pain: ↑↑ in lying down and ↓↓on sitting and leaning forward.
2- Sudden onset of D.M. in 25% of cases.
3- Slight jaundice, usually late, and only in 10% of cases.
4- Anorexia, Asthenia & Anaemia.

Signs:

They are usually few and reflect complications.

Investigations:

I- Laboratory :

1. Stool analysis: steatorrhea + occult bl. in periampullary type

2. Liver function tests: .S. bilirubin (direct type), and alkaline ph. enz.

3- Tumour markers: CA 19-9 & CEA are usually elevated

II- Imaging studies:

1. U/S, CT & MRI: to confirm diagnosis and assess operability .

2. PTC & ERCP. : With cytological examination of pancreatic juice.

3. Coeliac & Sup.Mes. angiography: may detect small tumours

Differential Diagnosis:

From other causes of jaundice specially calcular obst. Jaundice.

I- Operable cases:

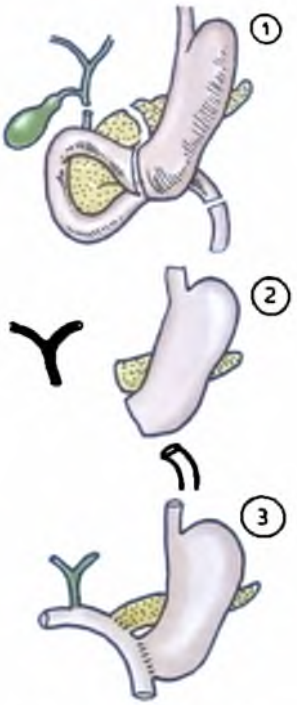
- Pre operative preparation (see obst. jaundice).
- Operation:

A- Cancer head: Pancreaticoduodenectomy (*Whipple's op.*)

- We remove
  1- Pancreatic head within the duodenal loop and pylorus.
  2-The gall bladder, CBD and distal part of CHD.
- Then anastomose the proximal jejunum to the proximal CHD, distal pancreas and stomach (figure 1, 2, 3).
- Some surgeons prefer to save the pylorus (Pyloric saving Whipple), others prefer to anastomose the pancreatic body to the stomach (pancreatico-gastrostomy)

B. Cancer body and tail : distal pancreatectomy.

We remove the body and tail of pancreas + splenectomy.



Whipple's operation

II- Inoperable cases: palliative measures:

a- Surgical bypass: cholecystojejunostomy + jejuno-jejunostomy to avoid reflux of food in biliary tree that may cause cholangitis.

b- Endoscopic stenting of CBD with metal stent.

c- Pain: chemical splanchenectomy .

Cholecystojejunostomy + jejuno jejunostomy

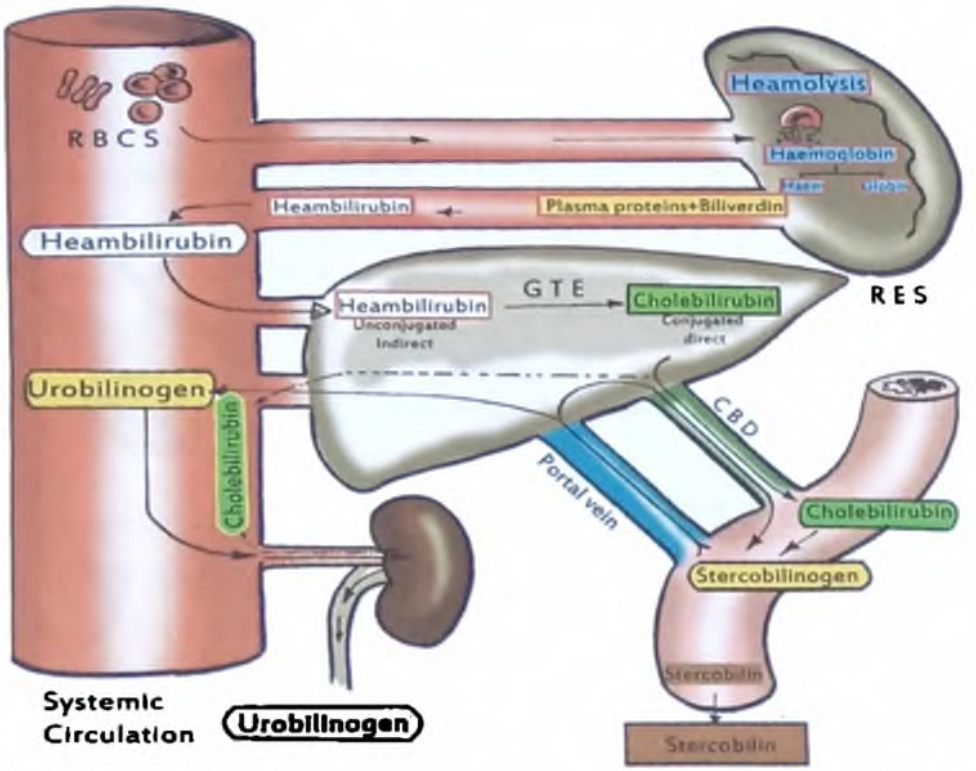
Prognosis:

Very bad.

Jaundice

Definition:

Yellow discoloration of skin, mucous membranes & sclera due to rise in serum bilirubin >2mg %



RBCs = red blood corpuscells

GTE = Glucoronyle transferase enzyme

RES = reticuloendothelial system

CBD = common bile duct

Normal steps of bilirubin metabolism

1- Production of bilirubin:

Haemolysis of old RBCs occurs in reticulo-endothelial System with liberation of Hb. which lastly gives rise to bilirubin, which is carried on plasma protein as haembilirubin, unconjugated bilirubin, or indirect bilirubin.

2- Uptake of bilirubin in the liver:

Haembilirubin is taken up from circulation by hepatocytes.

3- Hepatic conjugation of bilirubin:

Bilirubin is conjugated in the liver with glucouronyl group by glucouronyl transferase enzyme to be transformed into cholebilirubin , direct or conjugated bilirubin.

4- Excretion of cholebilirubin through the biliary tree:

Cholebilirubin is then excreted through bile ducts and CBD. to the intestine where, part is reabsorbed to the liver through the portal vein and then re-excreted into biliary tree to the intestine (enterohepatic circulation). Small part reaches the systemic circulation to be excreted in the urine as urobilinogen. The major part reaches the intestines as stercobilinogen, which is oxidized by the colonic bacteria into stercobilin, which gives the stool its normal colour.

Aetiology:

I- Excessive production of bilirubin: (Step 1):

As in hemolytic conditions.

II- Reduced hepatic uptake of bilirubin: (Step 2) as in:

1-*Gilbert's disease*: a hereditary disorder due to deficiency of Y & Z protein responsible for transport of bilirubin.

2-Viral hepatitis.

III- Reduced hepatic conjugation: (Step 3) as in:

1-Physiological jaundice of newborn due to Immaturity of glucouronyl transferase enz.

2-*Crigler. Najjar syndrome*: It is congenital absence of glucouronyl transferase enzyme.

3-Hepatocelluler diseases.

IV-Reduced excretion of conjugated bilirubin : (Step 4):

Due to intra-hepatic or extrahepatic cholestasis.

Types:

1-*Retention jaundice*: due to retention of haembilirubin in the circulation. It may be due to excessive haemolysis (Haemolytic jaundice) (Step 1), reduced hepatic uptake (Step 2) OR reduced hepatic conjugation (Step 3).

2-*Regurgitation jaundice*: due to regurgitation of cholebilirubin into the circulation secondary to cholestasis (Step 4), either intrahepatic or extrahepatic (obstructive jaundice.).

3-*Retention regurgitation jaundice:* as occurs in viral hepatitis or long standing obstructive or haemolytic jaundice.

A. Haemolytic jaundice

Definition:

Jaundice due to excessive destruction of RBCS.

Aetiology: causes of haemolysis

I-Corpuscular causes or primary haemolytic anaemia:

1-Membranopathy: e.g. spherocytosis, and elliptocytosis.

2-Haemoglobinopathy: e.g., Thalassaemia, and sickle cell anaemia.

3-Enzymopathy: e.g. favism (G. 6.P.D. deficiency).

II-Extra corpuscular causes: secondary haemolytic anaemia:

e.g. drugs, poisons ,auto immune haemolytic jaundice & hypersplenism.

Pathology:

Excessive haemolysis → production of excess haembilirubin beyond the hepatic capacity for conjugation → retention jaundice and exaggerations of bilirubin metabolism → ↑↑ stercobilin in stool → dark stool & urobilinogen in urine (darkens on exposure to air for sometimes).

Thalassaemia major

Cooley's anaemia or Mediterranean anaemia

Definition:

It is hereditary haemolytic anaemia transmitted as autosomal dominant trait characterized by abnormal over production of Hb F.

Actiology

The defect lies in Hb synthesis (Haemoglobinopathy) with over production of Hb F with decreased survival of abnormal cells.

Incidence

It is prevalent in the Mediterranean region. . *(From Greek thalassa 'sea')*

Clinical picture:

-Marked anaemia, moderate jaundice, huge Hepato-splenomegaly.

-Mongoloid facies (overgrowth of bone marrow of facial bones), retarded growth, and leg ulcers.

-Mortality is related to Iron deposition in different tissues especially myocardium due to repeated bl. transfusions.

Investigations:

1-Bl. Picture: decreased Hb, RBCs with target cells & reticulocytosis.

2- Decreased osmotic fragility.

3- Increased Serum bilirubin and serum iron.

4-Electrophoresis: the investigation of choice (Hb F & Hb A)

Treatment:

1-Repeated blood transfusion to correct anaemia + iron chelation (Disferral).

2-Splenectomy: in cases with proved hypersplenism or huge spleen.

B- Hepatocellular Jaundice

Definition:

Retention-regurgitation jaundice due to failure of the liver to conjugate & excrete all bile pigments.

Aetiology:

1-Congenital: congenital syphilis and congenital toxoplasmosis.

2-Traumatic: a-*Physical:* severe burns.

b-*Chemicals:* chloroform, chloramphenicol, halothane...etc.

3-Inflammatory: viral hepatitis, infectious mononucleosis, & yellow fever.

4-liver cell failure: end stage of cirrhosis and prolonged obstructive Jaundice.

5-Neoplastic: liver tumours.

Pathology:

The Hb catabolism is normal, but, the liver cannot conjugate and excrete all the bile pigment so, part of unconjugated bilirubin is retained in the circulation and part of the conjugated bilirubin is regurgitated to the circulation (retention -regurgitation jaundice). The diseased liver cannot excrete the absorbed urobilinogen again so; it is excreted in urine in increased amount.

C-Chloestatic Jaundice

Definition:

It is regurgitation jaundice as the conjugated bilirubin regurges to the circulation due to ***BLOCK*** in its excretion mechanism.

Aetiology:

I-Intrahepatic cholestasis:

1-*Dubin Johnson Syndrome*: hereditary defect in excretion of cholebilirubin (not bile salts) into bile ductules. with black liver.

Rotor's syndrome is a variant of this syndrome but with absence of hepatic pigmentation.

2- Drug induced cholestasis: contraceptive pills, steroids, and phenothiazine.

3- Chloestatic type of viral hepatitis (Watson's syndrome).

4- Chloestatic jaundice of pregnancy.

5- Post operative chloestatic jaundice.

6- Primary biliary cirrhosis.

II-Extrahepatic cholestasis = Obstructive jaundice = Surgical jaundice:

1-Congenital: Congenital biliary atresia and choledochal cyst.

2-Traumatic: Haemobilia and postoperative stricture.

3-Inflammatory: -Acute and chronic pancreatitis, and pancreatic cysts.

-Acute cholangitis and sclerosing cholangitis.

4- Parasites: fasciola and ascaris

5-Choledocholithiasis (CBD. stones) 38%.

6-Malignancy: (54%):

a-Cancer head of pancreas & periampullary carcinoma.

b-Malignant lymph nodes at porta hepatis .

Pathology:

Both Hb catabolism and hepatic conjugation are normal, but, the passage of the conjugated bilirubin to the intestine is prevented, so, it regurgitates to the circulation and passes in urine giving it the characteristic dark color. In addition, bile salts regurgitate to the circulation leading to bradycardia and Pruritis. The stool becomes pale, bulky, greasy and offensive. (steatorrhea). Urobilinogen is absent in urine.

N.B. for obstructive jaundice see later.

Post operative jaundice

It is a common postoperative event with up to 20% overall incidences.

Causes:

1-Hemolytic:

-Absorption of haematoma e.g. retroperitoneal haematoma.

-Incompatible blood transfusion or excessive transfusion.

2-Hepatocellular jaundice:

-Overstress imposed to pre-existing liver pathology (operative-drugs-anesthetic).

-Accidental ligation of major blood supply to the liver (arterial or portal).

-Viral hepatitis (at least 2 months incubation period).

3-Cholestatic jaundice:

-Post operative chloestatic jaundice.

-Extra hepatic biliary tract. obstruction (stricture- missed stone -pancreatitis).

-Trans-peritoneal absorption of bilirubin after biliary leak.

Calcular Obstructive Jaundice

Definition:

It is regurgitation jaundice due to obstruction of major biliary tract by a gallstone.

Aetiology:

The gallstone in the biliary passage may be:

1-Migrating stone from the gall bladder (common).

2-Primary stone formed in the biliary tree (uncommon).

Pathology:

I-General: feature of chloestatic jaundice (see before).

II-Local: that of obstruction.

1-At the site:

The stone may be impacted in:

a-Supraduodenal portion of CBD. (commonest site) .

b-Retroduodenal portion of CBD. (2nd common).

c-At the ampulla of Vater (pregnant papilla).

d-Common hepatic or major hepatic duct (rare) .

2-Above the obstruction:

a-The biliary tree: dilated and contain biliary mud due to stasis →infection.

b-The Gall bladder: is usually contracted (80%) according to "*Courvoisier's law*".

However, it is dilated in about 20% of cases due to:

1-Cholesterol stone in healthy gall bladder.

2-Primary C.B.D. stone (in healthy gall bladder).

3-Stone impacted in Hartmann's pouch compressing the common hepatic duct from outside (*Mirizzi's syndrome*) or double impaction i.e. stone in the CBD & another in the cystic duct.

c-Liver:

-Dilated intrahepatic biliary radicles → hydrohepatosis→ biliary cirrhosis.

-If infection → suppurative cholangitis → multiple liver abscesses→ fibrosis.

-prolonged stasis → white bile (bad prognosis).

3-Below the abstraction:

The bile does not reach the intestine leading to:

a-Steatorrhea: The stool is pale bulky greasy & offensive.

b-Lack of absorption of fat soluble vitamins (A, D, E, K) .

c-Decrease in faecal stercobilin & bile salts & decrease in urinary urobilinogen .

Complications:

I-General:

1-Bleeding tendency: due to lack of absorption of fat-soluble vit K.

2-Acute renal failure: in long standing cases with bad prognosis.

3-Liver cell failure: due to biliary cirrhosis when combined with renal failure → hepato-renal syndrome (fatal).

4-Pruirtis: due to irritation of the skin by bile salts.

II-Local:

1-Infection ⟶ suppurative cholangitis & liver abscesses.

2-Hydrohepatosis & biliary cirrhosis.

3-Steatorrhea & nutritional deficiency.

Incidence:

It is common in 5F (**F**emale, **F**atty, **F**orty, **F**ilthy & **F**ertile).

Clinical Picture:

Symptoms:

The patient complains of *Charcot's Triad* (intermittent pain, fever & jaundice).

1- Intermittent pain, which is colicky in the epigastrium or right hypochondrium.

2-Fever is usually accompanied with rigors.

3-Jaundice is deep greenish yellow with dark urine and pale stool.

Signs:

1-Tenderness in the Epigastrium or right hypochondrium.

2-Murphy's sign & Boas sign are usually + ve.

3-The liver is often palpable and tender, but, the gall bladder is usually impalpable.

Investigations:

I-Laboratory:

1-Blood: increase in s.bilirubin , mainly the direct portion (direct Van Den Berg reaction).
Increase in the liver enzymes especially alkaline phosphates (> 30 K.A unit).

2-Stool: decrease in stercobilin (pale).
-increase in undigested fat (bulky & greasy).

3-Urine: Increased cholebilirubin (dark), decreased urobilinogen, and increased bile salts → frothy urine.

4-Prothromobin time: is prolonged in long standing uncontrolled cases.

II-Imaging studies:

1-Plain x ray may show the stone in a low percent of cases.

2-Ultrasound: of great help to show dilated C.B.D. & Intrahepatic biliary radicles dilatation (IHBR) and sometimes the stone.

3-Endoscopic Retrograde Cholangio Pancreatography (ERCP): It delineates the biliary passages and localizes the site of obstruction.

4-Percutaneous Transhepatic Cholangiography (PTC.).
It is also helpful but, invasive. It may be combined with ERCP.

ERCP

5- Magnetic Resonance Cholangio-Pancreatography (MRCP):
Now. It is the standard and the best method for imaging of the biliary tree. It does not need contrast material and gives a very good imaging data of the biliary system.

6-Computerized Tomography (CT) Scan: in difficult cases.

N.B. oral & intravenous cholangiogram are contraindicated in obstructive jaundice.

Differential Diagnosis:

From malignant obstructive Jaundice (see later).

Treatment:

I-Conservative treatment:

Indications:

1-Pre-operative preparation.

2- Early cases, which show improvement with these measures.

Measures:

1-High carbohydrate diet, low in fat.

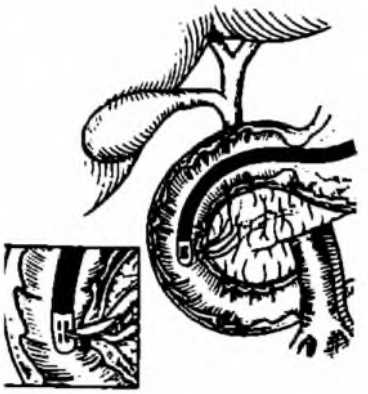
2-IV. fluids to maintain good renal perfusion and diuresis .

3-Mannitol 20% immediately pre-operative.

4-Vit K injection to guard against hypoprothrombinaemia.

5-Antibiotics to guard against secondary infection.

6-Observation.


Endoscopic papillotomy

- If the patient shows improvement, these measures are continued for at least 2 weeks. If pain, jaundice & fever were persistent or progressive, prepare for urgent surgery.

II-Interventional measures:

1-Endoscopic sphincterotomy (papillotomy) and stone extraction.

a-All causes should have a trial of stone extraction by *Dormia basket* during ERCP.

b-Cases unfit for surgery or missed stone after surgery .

2-Percutaneous Trans-hepatic Drainage (PTD).

For neglected cases, unfit for urgent surgery.

Choledocholithotomy

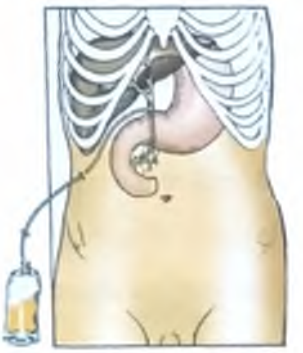
III-Operative treatment:

The classic operation is choledocholithotomy with cholecystectomy if possible. Choledocholithotomy means extraction of stone through supraduodenal or transduodenal approach.

After removal of stone, the CBD. is cheeked with Bake's dilators for stricture:

a- If there is stricture (low), do choledochoduodenostomy.

b- If not, close CBD. over a T tube to bypass the period of postoperative oedema and avoid jaundice & leakage.



Position of the T tube

After care of a T tube:

-It is clamped for increasing period between the 7th and 10th day.

-If there is pain, jaundice or leakage, do T tube cholangiogram to exclude missed stone or stricture.

Missed stone:

Missed stone is a stone detected in the biliary system within 6 months after biliary surgery

Management

1- Saline washout: with success rate about 20%.

2- Heparinized saline washout: 25000 IU in 250 cc saline /8h for 5 day (30% success).

3- Cholesterol solvents: e.g. Chenodeoxycholic acid, Monooctanoin, MTBE (Methyl Tertiary Butyl Ether) with 60% success.

4- Dormia Basket extraction: passed through the track of the T tube, which is left for 4-6 weeks (success rate 70-95%).

5- Endoscopic sphincterotomy & stone extraction.

6- Surgical re-exploration.

Removal of a missed stone with Dormia busket

Malignant obstructive Jaundice

Definition:

It is regurgitation jaundice due to obstruction of biliary passages by malignant growth.

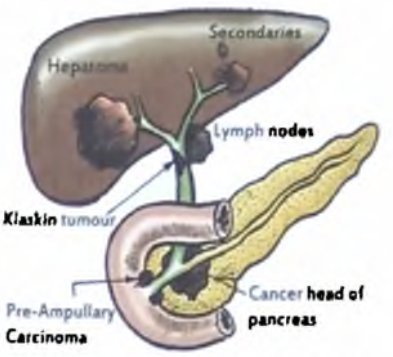
Aetiology: from below upward:

1-Perimapullary carcinoma & cancer head of pancreas.

2-CBD. carcinoma and Klatskin tumour (CHD cancer).

3-Malignant L.N. at porta hepatis.

4-Liver malignancy → 1ry: hepatoma, cholangio ca.
→ 2ry: (commoner).



Causes of malignant obstructive jaundice

Pathology:

1-At the site: the malignant growth.

2-Above the site: the same as calcular obst. jaundice with exception that the gall bladder is usually distended (Courvoisier's law) but occasionally, it is contracted due to:

a-Associated chronic cholecystitis with fibrosis .

b-The obstruction is high above the level of cystic duct (malignant LN. at porta hepatis).

3-Below the site: The same as calcular obst. jaundice.

Complications:

The same as in calcular type + spread of the tumour.

Incidence:

It is common in old males.

Clinical Picture:

Symptoms: "progressive *painless Jaundice*".

-Pain is usually absent or dull aching in the epigastrium & referred to the back.

-Progressive loss of weight with symptoms of complications.

Signs:

-The jaundice is deep greenish yellow & progressive.

-Progressive cachexia.

-The gall bladder is usually distended & palpable.

Investigations:

The same as calcular obst. jaundice + liver scan for secondary deposits..

Differential Diagnosis:

From other causes of jaundice especially calcular obstructive jaundice.

Item	Calcular obst. jaundice	Malignant obst. jaundice
History		
-Age & sex	Middle age female	Old male
-Onset & course	acute onset & intermittent course	gradual onset progressive course
-Pain	severe colicky in Rt. hypochond.	absent or dull aching in epigast.
Examination		
General: Weight loss	Slight	Marked
General: Oedema L.L.	Absent	May be (IV.C. obst)
General: Phlebothromb.	Absent	May be (Trousseau)
Abdominal: Liver	Enlarged smooth	May be nodular (2ries)
Abdominal: G.B.	Usually impalpable	Usually palpable
Abdominal: Ascites	Absent	May be (malignant
Investigations		
-Ultrasound -C.T & ERC.P.	detect the stone	shows the growth

Treatment:

According to the cause and its stage:

-Pancreatic carcinomas (see before).

-Higher obstruction is usually irresectable and choledocho-jejunostomy or even Percutaneous Transhepatic Drainage (PTD) relieves jaundice.

Prognosis: bad.

Courvoisier's law:

The GB is usually not palpable in calcular obstructive jaundice because it is the seat of chronic inflammation and fibrosis (shrunken) and usually palpable in malignant obstructive jaundice as it is healthy and distensible .

Exceptions of the law

I-Palpable G.B. in calcular O.J.	II-Impalpable G.B in malignant O.J.
1-Cholesterol stone in healthy GB.	1-Associated chronic calcular cholecystitis with cancer head.
2-Double impaction.	2-Malignant gland at porta hepatis.
3-Stone in Hartmann's pouch obst. Both cystic and CBD.	

How to investigate a case of jaundice?

[I] Clinical

A- History:-

1- The age of the patient

- Children & adolescence → haemolytic jaundice
- Young adult → hepatocellular jaundice
- Middle age → calcular jaundice obstruction
- Old age → malignant jaundice obstruction

2-Sex

- Male : malignant obstructive jaundice
- Female : calcular obstructive jaundice

3- Onset & course

- Acute onset and intermittent course → calcular obstructive jaundice
- Acute onset and regressive → viral hepatitis
- Acute onset and progressive course → cirrhosis , malignant obstructive jaundice
- Gradual intermittent course → haemolytic jaundice

4- Pain

- Severe colicky pain → calcular obstructive jaundice
- Dull aching epigastric pain referred to the back → malignant obstructive jaundice
- Right hypochondrial dull aching pain → viral hepatitis
- Bone aches → haemolytic jaundice
- Painless jaundice → malignancy

5- Urine & stool

- Very dark urine & pale stool → obstructive jaundice
- Dark urine & lightly coloured stool → hepatocellular jaundice
- Normally coloured urine & dark stool → haemolytic jaundice

6- Associated symptoms

- High fever (with rigors) & colic → calcular obstructive jaundice
- Mild fever & nausea → viral hepatitis
- Pruritis is more evident in obstructive jaundice
- marked weight loss → malignant obstructive jaundice

- 7- Past history
  - Past history of gall bladder problems or operations → obstructive jaundice
  - History of blood transfusion , drug abuse → viral hepatitis
  - History of bilharziasis , hepatitis or alcoholism → liver cirrhosis

8- Family history: It is positive in haemolytic jaundice

B- Examination

(1) Generally

1- The colour of the jaundice
  - Olive green colour → obstructive jaundice
  - Orange colour → hepatocellular jaundice
  - Lemon coloured → haemolytic jaundice

2- Bradycardia , itching markings →obstructive jaundice

3- Severe pallor due to anaemia → haemolytic jaundice

4- Manifestations of liver cell failure → hepatocellular jaundice.

5- Lower limb oedema → hepatocellular and malignant obstructive jaundice

6- Cachexia is more marked in malignant obstructive jaundice

7- Leg pigmentation and ulceration → haemlytic jaundice

(2) Abdominally

1- The liver is enlarged in most cases except in cirrhotic patients where it is shrunken

2- Spleen:
  - Mildly enlarged in viral hepatitis and 10 % of malignant obstructive jaundice
  - Moderately enlarged in haemolytic jaundice
  - Hugely enlarged in cirrhotic patients
  - Not enlarged in calcular obstructive jaundice

3- Gall bladder:
  - Not palpable in most cases
  - palpable in 80 % of malignant obstructive jaundice (Courvoisier's low)
  - some cases of calcular obstructive jaundice (exception of the law)

4- Ascites is present in cirrhotic cases and some cases of malignant obstructive jaundice

[II] Investigations

A- Laboratory investigations

1- Liver functions
  - High direct bilirubin (Direct Van Denberg reaction) with high alkaline phosphatase → obstructive jaundice
  - High indirect bilirubin (in direct Van Denberg reaction) → hemolytic jaundice

- High both direct & indirect bilirubin (biphasic Van Denberg reaction)with disturbed all liver function → hepatocellular jaundice

2- Stool analysis

- (dark) high stercobilin → hemolytic jaundice
- (pale) low stercobilin → obstructive and hepatocellular jaundice

3- Urine analysis:

- high cholebilirubin and bile salts → obstructive jaundice
- high urobilinogen → haemolytic jaundice
- high cholebilirubin and urobilinogen → hepatocellular jaundice

4- CBC: to diagnose haemolytic anaemia

B- Radiological investigation

1- Plain X ray rarely shows the shadow of stone

2- Oral & IV cholangiogram contraindicated

3- U/S, CT or better MRCP: they are very valuable, non-invasive investigations that give very important data especially in obstructive jaundice

4- ERCP: in obstructive jaundice, it is very helpful diagnostic procedure beside its therapeutic role

5- Percutaneous transhepatic cholangiogram (PTC), It is also helpful to diagnose obstructive lesions in the biliary system especially if ERCP fails

PERITONEUM & RETROPERITONEUM

Acute peritonitis

I-Acute generalized peritonitis.

Aetiology:

Causative organisms: Pyogenic organisms commonly E. coli, Streptococci, Pneumococci, and Staph. Aureus. Rarely, anaerobic organisms.

Routes of infection:

1-Direct: through penetrating wound.

2-Extension from inflamed organs: acute appendicitis, & acute salpingitis.

3-Leakage from rupture or perforated viscus e.g. perforated peptic ulcer, rupture gut, perforated diverticulitis, and rupture bladder.

4-Ascending infection: only in females through fallopian tubes.

5-Lymphatic spread from pleura.

6-Blood spread: from distant focus in pneumonia & septicemia.

(The commonest cause is perforated appendix then perforated peptic ulcer).

Pathology:

Types:

1-Localized Peritonitis: natural factors that help localization are:

a) Anatomical division of the peritoneal cavity into greater & lesser sacs. The former is divided into multiple compartments. This helps localize of infection to one compartment.

b) The greater omentum (policeman of the abdomen) is attracted, becomes adherent to the focus of infection and envelops it.

c) The reflex ileus and reflex rigidity immobilize both the intestine & abdominal muscles to help localization.

d) The fibrinous exudate glues the loops of the intestines together with omentum & abdominal wall to shut the infection off the rest of the peritoneal cavity.

2-Generalized (diffuse) Peritonitis:

Spread of infection through the peritoneal cavity:

A- High virulent organism or low resistance of the patient.

C- Dramatic onset (e.g. perforation) before localization takes place.

D- Persistent contamination (e.g. continuous leakage).

E- Prevention of localization by enema, and oral feeding.

Complications Causes of death:

1-Shock, Dehydration, and electrolytes imbalance.

2-Toxaemia, & septicemia.

3-Paralytic ileus.

4-Adhesive intestinal obstruction.

5-Residual abscesses (subphrenic , pelvic , paracolic…..etc.).

Clinical Picture:

Symptoms:

-History of the cause.

-General constitutional symptoms: Fever headache, anorexia & malaise

- Pain: varies in severity, but, usually severe and may be aching.
- Vomiting: early is a reflex, but later, it is due to ileus.
- Constipation: absolute.
- Distension: progressive.

} The famous tetrad

Signs:

General: Fever, tachycardia, late toxic facies, sunken eyes, and collapse.

Local abdominal signs:

Inspection: - The patient lies supine with flexed knee.
- Diminished abdominal respiratory movement.

Palpation: - Generalized abdominal tenderness, but maximum over site of cause.
- Abdominal rigidity.
- Rebound tenderness is pathognomonic.

Percussion - Tympanitic resonant abdomen (distended loops).
- Shifting dullness is late.
- Masked liver dullness in perforated gut (gas under diaphragm).

Auscultation -Diminished or absent intestinal sound.

Investigations:

1-Total leucocytic count. leucocytosis

2-Plain X-ray abdomen in erect position may show → Multiple fluid levels.
Gas under the diaphragm.

3-Ultrasonography: reveals free fluid.

4-Diagnositic peritoneal lavage → of pus, or intestinal contents.

Differential Diagnosis:

- From other causes of acute abdomen.

Treatment:

I-Preoperative measures:

1-Resuscitation: I.V. fluids.

2-Antibiotics with broad spectrum.

3- Nasogastric suction.

II-Operations = exploratory laparotmy:

-Aspiration of peritoneal exudate.

-Management of the cause e.g. appendectomy, closure of perforation etc...

-Peritoneal lavage.

-Closure of the abdomen with drains.

Prognosis:

It depends on many factors:

1-Primary cause: -Perforated appendicitis or peptic ulcer → MR = 15%.

-Perforated typhoid ulcer→ MR = 30%.

-Perforated diverticulitis → MR = 50%.

2-Age of the patient & his general condition.

3-Time before diagnosis and management: Prognosis is bad in neglected cases.

4-Concurrent medical diseases.

II- Localized peritonitis or Intraperitoneal abscesses

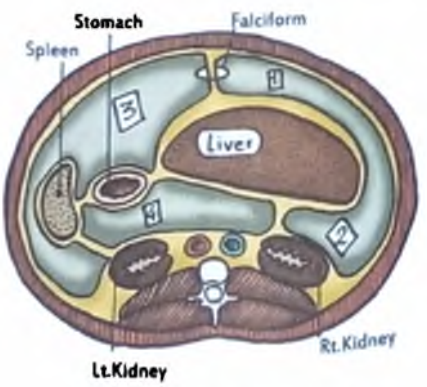
1-Subphrenic abscesses

Definition:

Accumulation of infected fluid in relation to the under surface of the diaphragm.

Aetiology:

Localization of generalized peritonitis in one of the subphrenic spaces, and each space has its causes.



Subphrenic spaces

Pathology:

Site: The supracolic compartment is subdivided by liver, stomach, spleen and their peritoneal attachments into:

I- Four Intraperitoneal spaces:

1-Rt. anterior intra-peritoneal:

- Between the Rt. lobe of the liver & diaphragm.

- Causes: Cholecystitis,perforated peptic ulcer, and liver abscess.

2-Rt. posterior intra-peritoneal = Morison or hepatorenal pouch (the commonest).

- Between the Rt. hepatic lobe and the Rt. Kidney.
- Causes:, perforated peptic ulcer, cholecystitis & acute appendicitis.

3-Lt. anterior intra-peritoneal:

- Between the Lt. Lobe of the liver, diaphragm & anterior abdominal wall.
- Causes: Post-splenectomy or gastrectomy operations.

4-Lt. posterior intra-peritoneal: (the lesser sac):

- Between the caudate lobe of liver, stomach and pancreas, Lt. Kidney.
- Causes: acute pancreatitis, and perforated posterior gastric ulcer.

***N.B.** Localization of generalized peritonitis is a common cause in all spaces.*

II- Three extraperitoneal spaces:

1-Rt. extraperitoneal space:

- It lies behind the bare area of the liver.
- Causes: acute retrocaecal appendicitis, liver abscess, and pyelonephritis.

2-3-The two perinephric spaces are considered subphrenic spaces.

- Causes: renal infection.

Clinical Picture:

The picture is that of suppuration of obscure origin.

(Pus somewhere, pus nowhere else, pus is there, under the diaphragm).

Symptoms:

1-History of the cause e.g. appendicitis, operation....etc.

2-Hectic fever, *(From Greek hektikos 'habitual')*& toxaemia.

3-Hiccup with pain referred to the shoulder (diaphragmatic irritation).

Signs:

General: Temperature is high, Tachycardia, & Toxic facies.

Local:

Inspection: -Diminished respiratory movements on the affected side.

Palpation: -Tenderness over the lower ribs on the affected side.

-Upper abdominal rigidity on the affected side.

Percussion: -Dullness over the base of lung on the affected side (effusion).

-In gas containing abscess, there are four percussion zones.

a-Normal lung resonance .

b-Dullness of the effusion .

c-Resonance of gas in the abscess .

d-Dullness of the liver .

Auscultation: Diminished air entry on the lung base.

Investigations:

1- Total leucocytic count is high.

2- Plain X-ray in erect position shows: * Sympathetic pleural effusion.

* Gas under diaphragm with fluid level.

* Elevated thickened fixed copula of diaphragm.

3- Ultrasonography is very helpful.

Treatment:

I-Conservative: Ochsner Sherren measures.

II-Interventional: closed drainage under U/S using special catheter (Apocath needle).

III-Surgical:

- Extra-serous drainage (extraperitoneal, extrapleural) to avoid contamination of both cavities.

* Anterior abscesses are drained anteriorly and posterior abscesses are drained posteriorly (may need resection of the last rib).

2-Pelvic abscess

Aetiology:

1-Acute pelvic appendicitis and acute salpingitis.

2-Localization of diffuse peritonitis in the pelvis.

Clinical picture:

Symptoms: -1-Deep-seated pelvic pain with mild toxemia.

2-Diarrhea with tenesmus and passage of mucous in stool.

Signs: 1-Mild suprapubic tenderness.

2- PR: tense tender cystic mass.

Investigations:

Ultrasonography is of great help.

Treatment:

I-Conservative: Ochsner- Sherren measures.

II-Extra peritoneal drainage through:

1-Posterior colpotmy or proctotomy if pointing occurred to vagina or rectum respectively.

2-Suprapubic approach.

Tuberculous Peritonitis

Aetiology:

Causative organism: Mycobacterium Tuberculosis.

Route of infection: always secondary:

-Direct spread: from TB. Enteritis, LNs. or salpingitis .

-Blood spread: from distant focus e.g. pulmonary TB.

-Lymphatic spread: from pleura or intestine (rare).

Pathology:

Types

1-Acute TB. peritonitis: The peritoneal cavity is filled with straw -coloured exudate with multiple tubercles over peritoneum and omentum.

2-Chronic T.B peritonitis: There are *four* pathological types:

1-Ascitic type: The commonest type. There are no adhesions.

The greater omentum is rolled up into sausage shaped mass straw coloured.

2-Encysted type: more common in females:

Localized cystic mass (DD. Ovarian cyst or mesenteric cyst).

3-Adhesive type: extensive adhesions may lead to intestinal obstruction.

4-Caseous type: The rarest form in patients with low resistance. Huge cold abscess forms and points anteriorly near umbilicus.

Incidence:

More common in children and young adults in undeveloped areas.

Clinical picture:

Symptoms:

1-TB. Toxaemia: night fever & sweat, loss of weight. etc.

2-Symptoms of the primary site: e.g. pulmonary TB.

3-Recurrent attacks of intestinal colic and vomiting.

Signs:

1- Mild tenderness and rigidity over the abdomen.

2- A sausage shaped mass may be felt above umbilicus (ascitic type).

3- A cystic intra-abdominal mass may be felt in encysted type.

4- Signs of complications e.g. fistula, cold abscess – etc.

Investigations:

1-Bl. Picture. Tuberculin test and chest X ray.

2-Ultrasonography: and U/S guided aspiration:

For → Biopsy for physical & chemical analysis.
→ Direct staining with Z/N stain.
→ Culture on Lowenstein Johnson media.

Differential Diagnosis:

1-Acute form from acute septic peritonitis (impractical).

2-Chronic ascitic type from mesenteric lymphadenitis, and intussusception.

3-Chronic encysted type from ovarian or mesenteric cyst.

Treatment:

I-Acute form:

-Laparotmy: evacuation of exudate with sampling.

-Sampling of omental tubercles for histopathology.

-Closure of the abdomen without drain.

-Antituberculous drugs.

II-Chronic form:

A-Conservative ttt:

-Antituberculous drugs, good nutrition, and avoidance of stress.

B-Surgery is indicated in:

-Ascites, drained by abdominal incision (not tapping), then closure without drains.

-Intestinal obstruction: Adhesions are usually marked so, lateral (side-to-side) anastomosis may be all what can be done.

-Intestinal fistula: resection of the fistulous loop and resection anastomosis.

Abdominal trauma

Aetiology = types

1- Blunt trauma.
2- Penetrating trauma. } See the wounds
3- Blast injury.

Clinical picture:

General: Signs of shock (pallor, tachycardia, and hypotension).

Local: - Ecchymosis, abrasions or even penetrating wounds of abdominal wall.
- Generalized tenderness & guarding maximum over the site of trauma.
- Abdominal rigidity raises suspicion of perforated gut.
- Shifting dullness may be elicited.
- Other signs specific to the injured organ e.g. Balance's & Kehr's signs.

Investigations:

1- Plain X ray in erect position: gas under diaphragm in ruptured gut.

2- Ultrasound (FAST): detects solid organ injury, free fluid in peritoneal cavity or retroperitoneal haematoma.

3- CT & MRI:. It is more accurate but not suitable for haemodynamically unstable patients

4- Selective angiography: in difficult cases.

5- Abdominal paracentesis: or better diagnostic peritoneal lavage (DPL): 20ml/kg saline is instilled by a drip set through small catheter inserted into the peritoneal cavity under local anaesthesia. The patient is then rolled on both sides and then the fluid is left to drain back. Frank bloody, blood stained or bile-stained fluid indicates internal haemorrhage or gut injury.

Treatment:

I- Resuscitations: I.V. fluids. blood transfusion ...etc.

II Exploration: manage accordingly.

1- Spleen, liver, see before.

2- Stomach: sutured in two layers.

3- Small intestines, right colon:– If small →suture in transverse manner in two layers .

–If large → resection anastomosis.

4- Lt. colon → suture + proximal colostomy.

5- Mesentry: -longitudinal tear → suturing.

-Transverse tears → resection anastomosis of the related loop of intestine.

6- , kidney (see later)

ACUTE ABDOMEN

Definition:

It is an acute abdominal pain that needs urgent relief.

***Aetiology*: Causes = D.D.(6 X 6)**

1-Chest causes:

1) Basal pneumonia.
2) Pleurisy.
3) Myocardial infarction.
4) Heart failure. (Congested liver).
5) Fracture ribs.
6) Pneumothorax.

2-Upper abdominal causes:

1) Perforated peptic ulcer.
2) Acute gastroduodenitis.
3) Acute cholecystitis.
4) Acute pancreatitis.
5) Acute hepatitis & liver abscess.
6) Internal haemorrhage.

3-Lower abdominal causes

1) Acute appendicitis (commonest).
2) Acute Crohn's disease.
3) Acute diverticulitis (Meckel's, colonic).
4) Intestinal obstruction.
5) Peritonitis (all causes).
6) Acute mesenteric lymphadenitis.

4-Pelvic Causes (Gynecological):

1) Rupture follicle (mittlschmertz).
2) Torsion ovarian cyst.
3) Rupture ectopic pregnancy.
4) Acute salpingitis.
5) Tubo-ovarian abscess.
6) Pelvic inflammatory disease (PID).

5-Retro peritoneal & spinal causes:

1) Ureteric colic.
2) Acute pyelonephritis.
3) Retroperitoneal haematoma.
4) Herpes Zoster (radiculitis).
5) Pott's disease.
6) Secondaries in the spine.

6-General (medical) causes

1) Diabetic abdomen (Ketoacidosis)
2) Tabetic crises in syphilis.
3) Familial Mediterranean fever
4) Henoch-Schoenlein purpura.
5) Acute porphyria
6) Uraemia.

Retroperitoneal Tumours

I- Primary retroperitoneal tumours arising from connective tissue:

1-Lipoma: sometimes turns malignant.

2-Sarcomas.

3-Teratoma and fibroma.

II- Tumours arising from retroperitoneal structures:

1-Lymph node: → Primary: Hodgkin and non-Hodgkin lymphoma.
↘ Secondaries: metastases.

2-Kidney: Hypernephroma & Wilm's tumours.

3-Suprarenal gland and sympathetic chain:

Ganglioneuroma, neuroblastoma, and pheochromocytoma.

Clinical Picture:

-Intra-abdominal mass:

*Early resonant abdomen (pushes the intestine in front).

*Fixed to posterior abdominal wall.

-Symptoms and signs of complications e.g. I.V.C. obstruction, metastasis, & malignant ascites.

Investigations:

1-Ultrasonography: inform about site, size and nature of the tumor.

2- C.T. scan & magnetic resonance imaging (MRI): *the gold standard.* It helps to study relations, vascularity, invasion of surrounding structures, and visibility of resection

3- Exploratorylaparoscopy or laparotomy:

-It may be needed for biopsy or resection if possible.

Treatment:

According to the nature and stage of the tumour.

Only about 20% of retroperitoneal tumour is resectable.

* * * * * * *

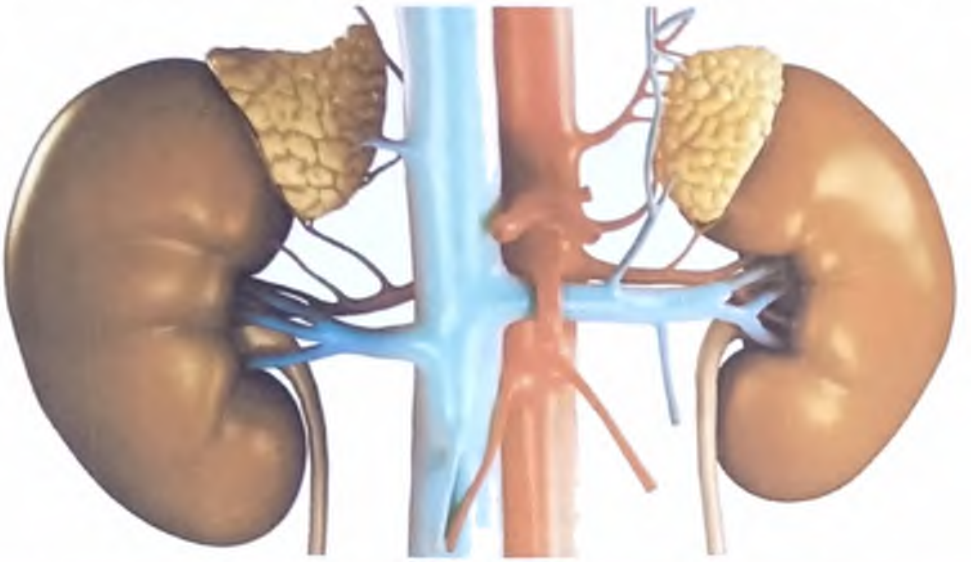
Adrenal gland

Embryology

develops from ectodermal cells migrating from the neural crest so it is while the cortex is developed in situ from mesoderm of intermediate cell mass.

Anatomy

,lands are two asymmetrical glands named so due to their intimate relation to the of the kidneys. Each weights 4-6 gm. The left is a little larger than the right.



The right adrenal gland :	The left adrenal gland :
It is pyramidal in shape.	It is semilunar in shape.
It does not reach the hilum of the right kidney.	It reaches the hilum of the kidney.
Its hilum is directed upward.	Its hilum is directed downward.
Its medial border is in close relation to IVC	Its medial border is to the left of the celiac ganglion.

Blood supply

A- Arterial : The gland has three arteries :

1- The superior suprarenal artery from the inferior phrenic artery .

2- The middle suprarenal artery from the abdominal aorta.

3- The inferior suprarenal artery from the renal artery.

B- Venous : The gland usually has one vein :

1- On the left side it drains into the left renal vein.

2- On the right side it is short (2-5 mm) and drains into inferior vena cava.

Lymphatic drainage:

Lymphatics from the adrenal gland drain into para-aortic LNs to the post. mediastinum LNs.

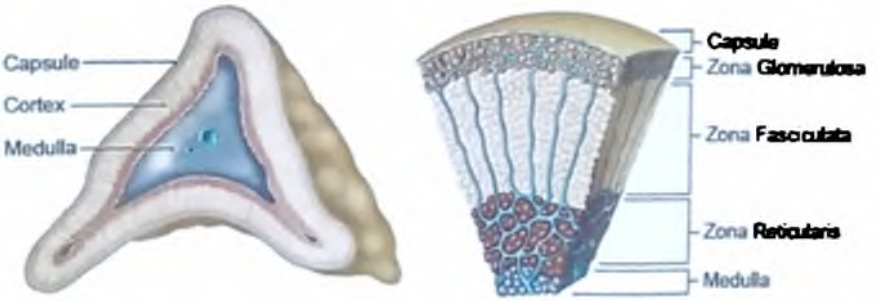
Nerve supply:

Mainly, by preganglionic sympathetic fibers from the celiac and renal plexus and They synap directly with the medullary cells (the adrenal medulla is a modified sympathetic ganglion).

Applied anatomy

- Being developed from the neural crest, the adrenal medulla cells are amine precursor uptal and decarboxylation (APUD) cells, so tumours of adrenal medulla e.g. pheochromocyton may be part of Apudoma or MEN (multiple endocrinal neoplasia) syndrome .
- Although, they are anatomically closely related, the kidney and adrenals developed from tw different embryonic origins, so if the kidney is ectopic or ptotic, it is not usually associate with displacement of the adrenals. However, fusion of the kidneys is usually associated wit fusion of the adrenals.
- The right renal vein is very short 2-5 mm or even shorter and it difficult to ligate wit considerable risk of avulsion from inferior vena cava so the right adrenalectomy is mor difficult than left one.

Histology



Physiology

Adrenal hormones

*The adrenal codex secrets:

1- mineralocorticoids e.g. aldosterone from zona glomerulosa

2- Glucocorticoids e.g. cortisole from zona fasiculata.

3- Sex hormones.g. androgen and oestrogen from zona reticularis.

*The adrenal medulla secretes:

- Catecholamines e.g. adrenaline and noradrenaline

Adrenal tumours

I- Adrenal cortex tumours .

A- Non functioning tumours .

B- Functioning tumours .

1- Cushing's syndrome .

2- Conn's syndrome .

3- Adrenogenital syndrome .

II- Tumours of the adrenal medulla.

A- Tumours of the sympathetic neurons .

- Benign : ganglion neuroma .
- Malignant : neuroblastoma .

B- Tumours of chromaffin cells:

- Pheochromocytoma .

Cushing's syndrome

Aetiology :

I- ACTH dependent Cushing's

1- Pituitary adenoma in two thirds of cases it is due to ACTH secreting adenoma (basophile adenoma) of the pituitary.

2- Ectopic ACTH production from bronchial adenoma or carcinoma or pancreatic islets tumours

II- Non ACTH dependent

- Adrenal tumours: Adrenal cortical adenoma in 20% and adenocarcinoma in 5% of cases are the cause of Cushing's syndrome.

Incidence

It is more common in females (♀: ♂ = 3:1) usually between 15 : 30 years

Clinical picture

1- Truncal obesity with a roll of fat at the back of the neck (buffalo hump) while the limbs are thin giving the patient the appearance of " *a lemon on match stick* "

2- Moon face and obliteration of supraclavicular fossae .

3- Thin skin with bad wound healing, easy bruisbility & abdominal striae.

4- Hypertension and amenorhea, loss of libido and hirsutism .

5- Osteoprosis and muscle weakness.

Investigations

I- Laboratory

[1] Estimation of plasma cortisone. level: loss of diurnal variations of cortisone is an early laboratory finding in Cushing's disease. Later on, high levels are detected.

[2] Low dose dexamethazone test: 1 mg is taken at night and plasma of cortisone is measured at the morning if it is low the diagnosis is in doubt. If it is high. Cushing syndrome is diagnosed.

[3] Estimation of plasma ACTH: It is high in pituitary & ectopic cases and low in adrenal cases.

[4] High dose dexamethazone test: 8 mg dexamethazone /day is given for 2 days then 17–hydro-corticosteroids (end product of cortisol) is estimated in urine. If they are reduced, pituitary Cushing is diagnosed, if not, ectopic sources of ACTH are suspected.

II- Imaging studies: (to locate the tumour).

2) Plain X ray of the chest.

3) CT scan of the head, chest and abdomen.

Treatment:

[1] Adrenal cases are treated with adrenalectomy of the diseased side. Postoperative corticosteriods (tapered gradually) are needed to avoid Addisonian crises as the contralateral adrenal is suppressed.

[2] Pituitary Cushing's syndrome is treated by

a) Hypophysectomy or microsurgical excision of the adenoma through trans-sphenoidal route.

b) Irradiation of the pituitary by implantation of yttrium 90 .

c) Bilateral adrenalectomy (not done nowadays)

[3] Ectopic ACTH secretion is treated by excision of the bronchial or pancreatic tumours if possible.

Prognosis:

Most cases survive 20 years after successful resection of adrenal adenoma, but in case of carcinoma, survival beyond 5 years is rare.

Hyperaldosteronism

Aetiology:

I- Primary hyperaldosteronism (Conn's syndrome): due to

1) Adrenal adenoma.

2) Bilateral adrenal hyperplasia.

3) Adrenal carcinoma (rare)

II- Secondary Hyperaldosteronism: due to

1) Liver disease: decreased metabolism of normally secreted aldosterone.

2) Renal ischaemia: due renal artery stenosis, polycystic kidney, hydronephrosis, coarctation of aorta..etc activates renin- angiotensin system that stimulates aldosterone secretion .

Incidence:

Conn's syndrome represents about 1-2% of all hypertensive patients.

Clinical picture:

1) Muscle weakness, polyurea, polydypsia .

2) Arrhythmia, hypertension which may be malignant causing stroke .

3) Tetany with carpo-pedal spasm and paraesthesia due to hypokalaemia.

Investigations:

I- Laboratory:

1) Blood chemistry: hypokalaemia, hypernatraemia and mild alkalosis.

2) Plasma renin is moderately suppressed in micronodular hyperplasia and markedly suppressed in adenoma while in secondary hyperaldosteronism, plasma renin is high.

3) Plasma aldosterone level is high. In adenoma, it is suppressed by simple intake of potassium or plasma volume expansion but in hyperplasia. It may be suppressed by cortisol or volume expansion.

II- Imaging studies (locating the tumour)

1) Abdominal U/S can detect tumour larger than 2cm.

2) CT scan of the abdomen (90% accuracy)

3) Adrenal scanning (scintigraphy) with **NP-59** to differentiate between adenoma which gives hot spot with suppression of the contralateral gland, carcinoma which does not concentrate the isotope with also contralateral gland suppression and hyperplasia that gives prominent bilateral image .

Treatment:

I- Conservative treatment:

Some cases are controlled with medical treatment as Spironolactone or amiloride, beside correction of electrolytes disturbance and control of hypertension.

II- Surgical treatment:

1) Adrenal adenoma is treated by unilateral adrenalectomy.

2) Adrenal hyperplsia is treated by right total adrenaleectomy and left partial or subtotal adrenalectomy (leaving only 30% of the gland). The left side is chosen for partial adrenalectomy because if recurrence occurs re-exploration is easier than the right side where inferior vena cava is at risk of injury.

Adrenogenital syndrome

Adrenogenital syndrome may present at three different age groups in three different forms.

I- Congenital form (congenital adrenal hyperplasia)

It is due to congenital enzymatic deficiency that leads to failure to synthesize cortisone with production of an intermediary steroids which have androgenic effect that cause musculinization

of genitalia e.g. *female pseudo-hermaphrodite* or *male macrogenitosomia precox.* ACTH is very high

II- Virilizing tumours:

- They are androgen secreting tumours of the adrenal they occur commonly around puberty..
- It causes precocious puberty with short stature, muscular hypertrophy, hirsuitism *(infant Hercules)*
- Enlargement of the penis or clitoris otherwise the external genitalia are normal.

III- Feminizing tumours:

- They are estrogen secreting tumours and they are rare.
- They mainly present with gynaecomastia, loss of libido, impotence.
- The tumour is usually highly malignant and large enough to be palpated in about 50% of cases.

Investigations

I- Laboratory .

1- Plasma cortisole is low and ACTH is high in congenital cases.

2- 17-Ketosteroids in urine are high in virilizing tumours.

II- Imaging studies: to localize the tumour

As before.

Treatment:

- Cortisole replacement is essential for congenital cases then plastic reconstruction for the genital organs.
- Excision of the tumour e.g. adrenalectomy for adult cases whenever possible.

Pheochromocytoma (PHEO)

It is a tumour that arises from chromaffin cells **10%** are malignant.

Actiology:

- About **10%** of the tumours are familial. Genetic predisposition is well known through MEN IIa and MEN IIb syndrome where PHEO is commonly associated with parathyroid and thyroid tumours. The offending gene has been located on chromosome **10.**

Pathology:

Site: - 90% of the tumours are from adrenal medulla.

- **10%** of the tumours arise from extra-adrenal sites like

1-Paraganlion retrogastric and retopancreatic.

2-Organ of Zuckercandle at the aortic bifurcation & around the origin of inferior mesenteric artery

3-Mediastinum, urinary bladder.

In **10%** of cases the tumours is multiple. Bilaterality and multiplicity are common in familial cases.

MAC: The tumours varies in size but usually small < 3cm in diameter, reddish brown in colour, vascular and may be solid or multicystic.

MIC: -The cells vary in size and shape with granular cytoplasm, oval nuclei with prominent nucleoli

- Areas of haemorrhage & necrosis and vascular and capsular invasion are often present even in benign tumour.

Complications: (causes of death)

1) Paroxysms of hypertension that may lead to cerebral stroke
2) Arrhythmia, cardiomyopathy, heart failure and infraction
3) Diminished blood volume and renovascular hypertension
4) Hypertensive retinopathy, retinal detachment and optic atrophy

Incidence:

- It commonly affects in young adult or middle age (both sexes), however **10%** of cases are children.
- The familial type is more common in children, with female predilection.

Clinical picture:

- A common complaint is fear. The patient says that *" I though I was going to die "*
- The main presentation is hypertension which may be persistent or paroxysmal
- Headache, vomiting, palpitation, dyspnea with attacks of anginal pain.
- Sudden death may be the first presentation.

Investigations:

I- Laboratory :

1- Estimation of free catecholamines and vanillyl mandelic acid (VMA) in urine patients with PHEO usually excrete free catecholamines > 100 mg and VMA > 7 mg in 24 hours urine collection

But there is **10%** false negative results also false positive results may occur.

2- Measuring the plasma catecholamine level is now possible with accuracy.

Patients with PHEO usually have figures higher than **1000** µg/ml

II- Imaging studies: to localize the tumour

1- Abdominal U/S, has 80-90% accuracy.

2- CT scan and MRI have accuracy approaching 100% they are also accurate in determining resectability and extent of the tumour.

3- Adrenal scan with (MIBG) is very helpful in localization of adrenal and extra-adrenal PHEOS also it can detect occult metastases.

Differential diagnosis

Symptoms of PHEO may simulate those of hyperthyroidism, diabetes mellitus and carcinoid syndrome.

Treatment:

I- Pre operative preparation

Patients with PHEOs should be adequately prepared in ICU for 1-3 weeks prior to surgery as follows

- We begin with α blockers such as phenoxybenzamin till mild orthostatic hypotension occurs then, beta blockers as propranolol are added 3-7 days before operation to control arrhythmias.

  Excess fluid therapy is added to expand the circulation and restore normal blood volume

Operation

1- Premedications such as pethedine and scopolamine are permitted but not barbiturates.

2- Nitrous oxide with oxygen is ideal but not halothane because it is arrythmogenic.

3- IV. infusion of alpha and beta blocking agents are given during surgery.

4- Continuous monitoring of pulse, bl. pressure, CVP & urine output is very importance.

5- All needed medications such as sodium nitroprusside, phentolamine, noreepinephine, lignocaine & propranolol should be ready to control any problem.

6- Generous anterior transperitoneal approach is favoured to permit adequate exploration, minimize manipulation of the tumour and allow ligation of the adrenal vein early as a first step.

7- Avoid undue manipulation, rupture or needle aspiration of the tumour as these may cause recurrence of the tumour even those which appear benign.

8- Close and perfect postoperative monitoring is essential.

Prognosis

Good preoperative preparation had lowered the operative mortality from 80 to 3%.

Minimal invasive surgery (MIS)

Definition:

It is a new technology that aims at performing diagnostic or therapeutic goals with minimal anatomical, physiological and psychological trauma to the patients.

MIS includes a wide variety of procedures such as:

I- Laparoscopic procedures.

II- Thoracoscopic procedures:

III- Perivisceral endoscopic procedures:

1- Retroperitoneoscopy. 2- Mediastinoscopy.

3- Subfascial endoscopic perforator surgery (SEPS) in varicose veins.

IV- Arthoroscopic procedures.

V- Endoluminal endoscopy:

1- Upper and lower GIT endoscopy. 2- ERCP and endoscopic stone extraction.

3- Cystoscopy, ureteropyeloscopy 4- Sinoscopy and laryngeoscopy

5- Hysteroscopy 6- Bronchoscopy

VI- Endovascular surgery and angiography.

VII- Interventional radiology (Image assisted surgery): It includes

1. Image (e.g. U/S) guided biopsy or drainage
2. Percutaneous dilatation & stenting of bile duct strictures
3. TIPS in portal hypertension
4. Percutaneous nephrolithotomy (PCN).

VIII- Extracorporeal shock wave lithotripsy (ESWL).

IX- Venriculoscopy in neurosurgery.

X- Local tissue destruction: It includes

1. Laser surgery 2- Cryosurgery.

3- Percutaneous injection of ethanol into tumours 4- Radiofrequency

Laparoscopy

Nowadays, most of abdominal surgical procedures can be performed laparoscopically.

Technique:

1- Creation of pneumoperitoneum by insufflating CO_2 gas into the peritoneal cavity usually through Veress needle to the pressure of 12 –16 mmHg.

2- Introduction of multiple ports trocars into the abdomen. Through the first port, a telescope connected to camera-video system is introduced to visualize the intra peritoneal structures.
3- The long thin laparoscopic instruments can then be introduced to handle the tissue and perform surgical procedures under visual control. These instruments can be armed with diathermy, laser or other energy sources that facilitate the surgical procedures.
4- After completion of the procedures and ensuring haemostasis, the pneumoperitoneum is deflated and the laparoscopic ports are withdrawn.

Advantages of laparoscopic surgery = Reverse of disadvantages of open surgery

1- Smaller wounds with less tissue trauma, better cosmesis and less psychological impact on the patient.
2- No exposure of internal organs with minimal cooling and dryness of tissues.
3- Minimal handling and less trauma, to tissue and hence early return of bowel function.
4- There is less postoperative pain and less limitation of respiratory movements so less incidence of chest complications.
5- Very fewer wound complications e.g. infection and dehiscence.
6- Rapid recovery, short hospital stay and rapid return to work.
7- Reduced contact between the surgeons and the patient's blood or body fluids with less possible incidence of instrument injuries so decreasing the incidence of transmission of diseases e.g. serum hepatitis and AIDS to the surgeons

Disadvantages of laparoscopic surgery:

1- Lack of direct handling of tissues and tactile feedback, which is important for evaluation of the local pathology.
2- It needs experience which is slow to acquire so, it needs longer duration than open surgery but with learning curve, the time is markedly decreased.
3- Difficult control of intra-operative bleeding largely due to light absorption by the extravasated blood that obscures the visual field.
4- Still there is higher incidence of iatrogenic injuries in comparison to open surgery. Most of these complications are due to lack of experience & bad judgment.

Complications of laparoscopic surgery:

I- Complications related to gas insufflation and trocar insertion:

1- Gas embolism usually due to insertion of the needle into blood vessel or escape of CO_2 into an opened venous channel.
2- Visceral injury by the needle or trocars.

3- Respiratory compromise due to high intra-abdominal pressure that splints the diaphragm interfering with full expansion of lungs especially in patients with restrictive lung disease.

4- Post-operative shoulder pain due to peritoneal irritation with CO_2 It resolves within 48 h.

II- Complications due to use of the laparoscopic instruments:

1- Visceral injury with laparoscopic instruments.

2- Diaphragmatic injury causing tension pneumothorax.

3- The hot telescope may cause significant burn if it touches the viscera.

4- Electro-cautery (diathermy) injury to the gut that may manifest after long time 4-10 days with intestinal leakage and peritonitis.

Contra-indications of laparoscopic cholecystectomy:

I- Absolute contraindications:

1- Late pregnancy
2- Acute cholangitis
3- Suppurative peritonitis
4- Major bleeding disorders

II- Relative contraindications

1. Previous upper abdominal surgery
2. carcinoma of the gall bladder
3. Advanced liver cirrhosis
4. Minor bleeding disorders

The role of laparoscopy in general surgery:

Laparoscopy has a very wide spectrum of use in general surgery. The most common uses are:

I- Diagnostic Laparoscopy: It is commonly applied in

1) Abdominal trauma.
2) Acute abdomen.
3) Abdominal malignancy.
4) Staging laparoscopy in lymphoma.

II- Therapeutic role:

1- Laparoscopic cholecystectomy.
2- Laparoscopic CBD exploration & stone extraction
3- Laparoscopic appendectomy.
4- Laparoscopic colectomy & proctectomy
5- Laparoscopic vagotomy & GJ.
6- Laparoscopic fundoplication
7- Laparoscopic cardiomyotomy
8- Laparoscopic partial and total gastrectomy
9- Laparospic splenectomy
10- Laparoscopic groin and ventral hernia repair
11- Laparoscopic liver resection.
12- Laparoscopic adrenalectomy
13- Laparoscopic ovarian cystectomy
14- Laparoscopic oophrectomy and hysterectomy
15- Thoracoscopic cervical symathectomy
16- Laparoscopic lumber sympathectomy
17- Laparoscopic gastric banding
18- Laparoscopic Reux en Y & minigastric bypass
19- Laparoscopic sleeve gastrectomy
20- Laparoscopic varicocelectomy
21- Endoscopic thyroidectomy
22- Endoscopic breast lumpectomy

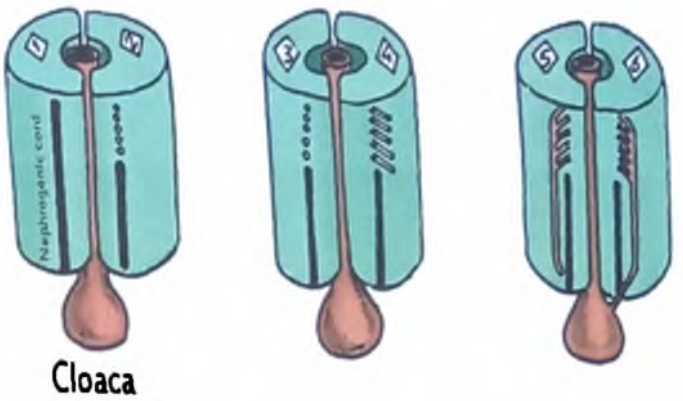
The Kidney

Embryology

-The kidneys develop from a mesodermal cord on the back of the coelomic cavity called the *nephrogenic cord.*They pass through three stages of development:

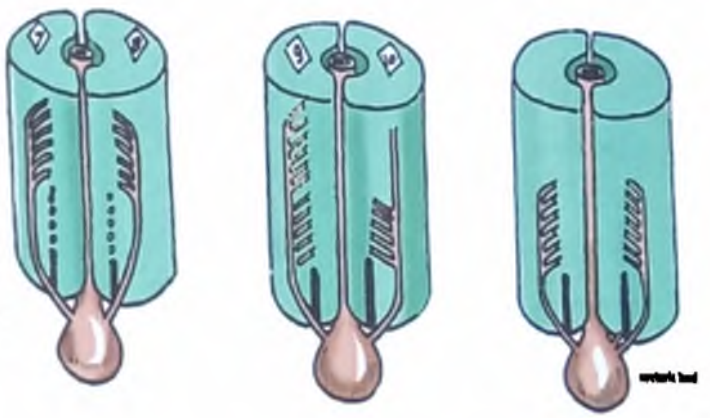
I-pronephros:

The cranial part of the nephrogenic cord is divided into multiple segments called *nephrotomes*. They then acquire a cavity and called *nephroceles*, that elongate laterally and then, caudally to fuse with each other forming a common duct called the*pronephric duct* that opens into the cloaca .



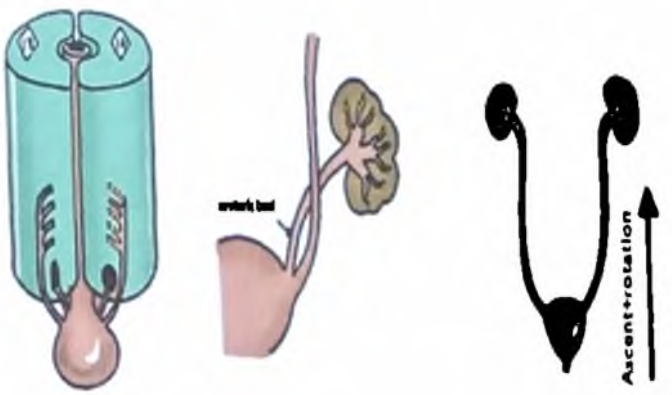
II-Mesonephros.

The middle part of the nephrogenic cord will undergo similar changes as before and as a result, *the mesonephric tubules* will open into the pronephricduct that change its name to the *mesonephric duct.*



III- Metanephros:

From the lower end of the mesonephric duct, *a ureteric bud* projects dorso-medially to touch the caudal end of the nephrogenic cord. The latter will form a cap *(metanephric cap)* .The ureteric bud then divides and branches to form calyces and the collecting system of the kidney ,while the metanephric cap will form the secretory system of the kidney.



Then, the two kidneys begin to ascend in the posterior abdominal wall to reach their final position in the loin. At first, the calyces are directed backward & medially .During ascent of the kidney, it rotates so that the calyces will be directed laterally.

Anatomy

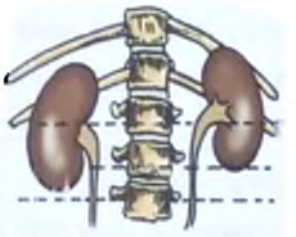
The kidneys are the vital organs for filtration of blood, excretion of waste products regulation of electrolytes and acid-base balance beside its endocrinal function.

Each kidney is about 10cm in length, 5cm in width & 2.5 cm in thickness.

Surface anatomy:

1- The Rt. kidney lies at a lower level than the Lt One, probably due to its relation to the liv

	Rt. kidney	Lt. kidney
Upper pole	11th intercostal space	11th rib
Hilum	Just below the transpyloric plane (L1)	Just [illegible] the transpyloric plane
Lower pole	Level of L3	Level of L2



Level of the kidneys

Morison's rectangle: from he back draw "

1- Two horizontal lines

-The first is at the level of the 11th thoracic

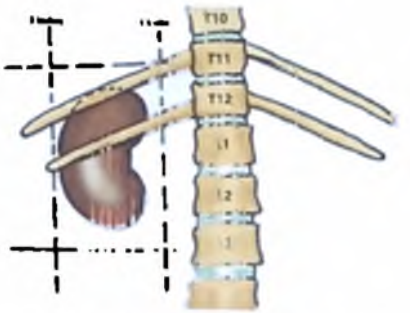
-The other is at the level of the third lumba

2- Two vertical lines

The first is one inch from the midline.

The other is three inches from the midline

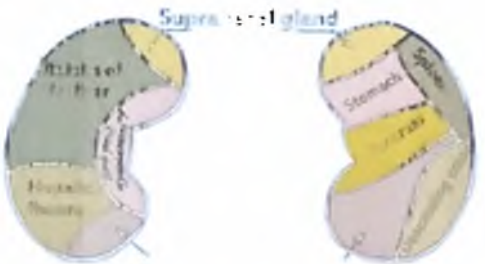
Draw the kidney in the rectangle

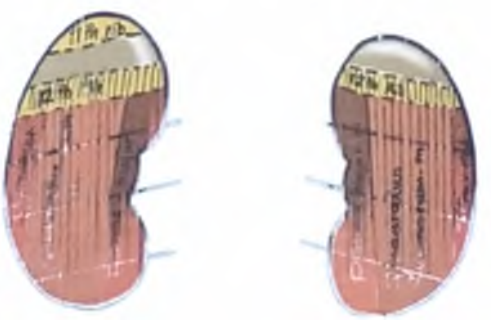


Morison's rectangle:

Relations:

the figures

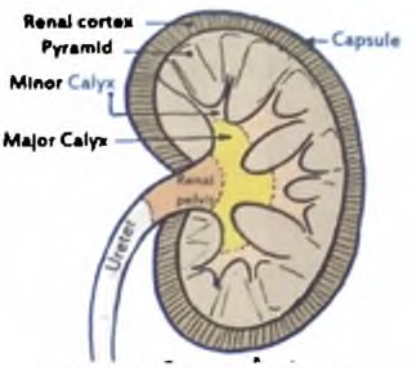




Internal appearance:

The kidney has an internal medulla and an external cortex that lies under the fibrous capsule.

The renal pelvis is divided into 2 or 3 major calices that divide into a number of minor calices. Into each minor calyx, project renal papilla from the apex of the renal pyramid.



Internal appearance

Capsules:

The Kidney has three capsules:

1- Proper fibrous capsule.

2- Perinephric fat (Fatty capsule).

3-Anterior fascia of gerota andRenal fascia (***Zuckerkandl's fascia***).

Blood supply:

Arterial: from the renal artery out of abdominal aorta at the level of L1.

Venous: Into the renal, vein which drains into inferior vena cava.

Nerve supply:

1- Parasympathetic: by branches from the coeliac plexus.

2- Sympathetic, from T12 & L1 splanchnic nerves.

Lymphatic drainage:

It drains into para aortic L.N.s.

Applied Anatomy

1- I.V.C. ascends in front of the Rt. renal artery. Also the Rt.renal vein is shorter than the Lt.one. Therefore, the right nephrectomy is more difficult than the Lt.nephrectomy because the Rt. renal pedicle is shorter than the Lt. one.

2- The renal artery is an end artery, and the kidney has a segmental blood supply. If a branch of the renal artery is occluded, a cone shaped segment of the kidney is infracted.

3- At the hilum of the kidney, the renal pelvis is the most posterior structure, the vein is the most anterior structure & the artery is inbetween. Therefore, during nephrolithotomy, we approach the renal pelvis from the back surface of the kidney

I- Congenital Anomalies.

I- Anomalies of number.

1- Unilateral renal aplasia.

2- Bilateral renal aplasia (incompatible with life).

3- Super numerary kidney.

II- Anomalies in size.

1- Hypoplastic kidney. 2- Hypertrophied kidney.

III- Anomalies of the site:

- Ectopic kidney.

IV- Anomalies of shape.

- Ox kidney (persist fetal lobulations).

V- Anomalies due to abnormal fusion.

1- S shaped kidney. 2- L.shaped kidney.

3- Discoid shaped kidney. 4- Horseshoe kidney.

VI- Anomalies due to failure of communication:

1- Solitary renal cyst. 2- Polycystic kidney.

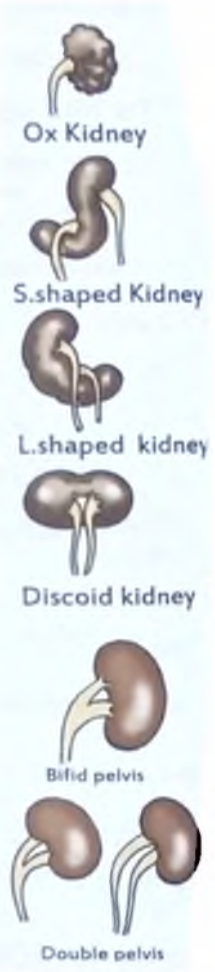
VII- Anomalies of the renal pelvis:

1- Bifid pelvis. 2-Double pelvis (with single or double ureters).

VIII- Anomalies of renal vessels:

1- Renal artery stenosis → renal hypertension.

2- Abrrent renal artery →hydronephrosis.



Ectopic Kidney

Aetiology:

Failure or abnormal migration of the kidney to its normal position.

Pathology:

Site:-Usually at a point in its pathway to the loin.

*In the iliac fossa (the commonest).

*In the pelvis (common).

- In crossed renal ectopia, the kidney crosses the midline to settle below the other kidney.

Complications

Ectopic kidney is more liable to

1-Trauma.2-Obstruction.3-Infection.4-Stone formation.

5-Pelvic kidney may be injured during labour or may obstruct it.

Clinical picture:

I-Asymptomatic cases:

Many cases are discovered accidentally during routine investigations.

II-Symptomatic cases:

1-Mass in the iliac fossa may by felt

2-Picture of complications e.g. infection, stone, obstructive uropathy.

Ectopic kidney

Investigations

1-Ultrasongraphy.

2-IVU shows the kidney in its abnormal position with straight ureter.

Differential diagnosis:

From mobile kidney.

	Ectopic kidney	Mobile kidney
Clinically	It cannot be pushed to the loin.	it can be pushed to the loin.
IVU.	-No difference in kidney position between erect & supine position.	-There is over mobility of the kidney between erect & supine position.
	-the ureter is short & straight	- Ureter is long & tortuous.
	-The renal artery arises from abnormal site.	-The art. arise from its normal site & level.

Treatment:

I- Asymptomatic cases:

Conservation and instructions to guard against violence e.g. Contact sports.

II- Complicated cases:

Only treatment for complications. Nephrectomy is not indicated as before.

Horseshoe kidney

Aetiology:

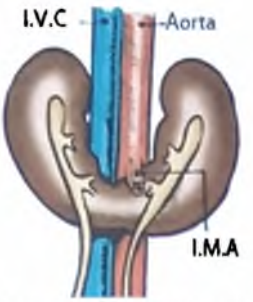
Abnormal fusion between the lower poles of both kidneys during their development in the pelvis.

Pathology:

Both kidneys are fused together at their lower poles with either connective tissue or true renal tissue (isthmus), giving a horseshoe mass. This fusion leads to the following:

* Failure of complete migration, as the kidneys are arrested at the level of L3 by the inferior mesentric artery that hooks over the isthmus.
* Failure of complete rotation especially the lower calyces which become malarotated i.e. (directed medially or posteriorly).

* The two ureters are near to each other, and descend over the isthmus then diverge downward, so, they are liable to obstruction.



Horse shoes kidneys

Complications:

1-Obstruction (stasis). 2- Stone formation.

3-Infection (sepsis). 4- Hydronephrosis.

5-Isthmus syndrome: due to compression of the ismuth on IVC. leading to oedema of the lower limbs with congestion pain. This pain increases with hyperextension of the spine ***(Rovsing's sign).***

Clinical picture:

I- Asymptomatic cases:

Most cases are asymptomatic and discovered accidentally during routine investigations.

II- Symptomatic cases:

1- Tender subumbilical retroperitoneal mass.

2- Complications e.g. Infection, stone & isthmus syndrome.

IVU

Investigations:

1- Ultrasonography & CT scan.

2- Intravenous (excretory) urography (IVU) shows:

1- Lower position of both kidneys.

2- Malrotation& approximation of calyces especially the lower ones.

3- Both ureters converge then diverge towards the pelvis giving *(flower vase appearance).*

Treatment:

I- Asymptomatic cases:

No treatment.

II- Complicated cases: Treatment of complication e.g. hydronephrosis, stone …etc.

-If one kidney is destroyed and the other is healthy, do heminephrectomy.

-Isthmus syndrome is treated with isthmotomy or symphysiotomy i.e. division of the isthmus.

Congenital polycystic kidney

Aetiology:

It is due to failure of communication between the secretory system (metanephros) and the excretory system, which develops from mesonephric ureteric bud.

It is a hereditary disease. It may be:

* Autosomal dominant (adult type).

* Autosomal recessive (infantile type).

Pathology:

Site: It is always bilateral, but one side is usually more affected.

Pathological changes:

-Both kidneys are studded with multiple cysts of varying size containing usually clear fluid.

-These cysts are closed cysts and not communicating with the pelvis or with each other.

-Some cysts would enlarge with time and compress the surrounding healthy nephrons causing their atrophy and finally renal failure.

- Associated splenic and hepatic cysts may exist. Also, liver fibrosis and portal hypertension.

Complications:

1- Haematuria, due to rupture of cysts into the pelvicalyceal system.

2- Polyuria: due to failure of the kidney to concentrate urine.

3- Hypertension, (75%): As renal ischaemia activates rennin - angiotensinogen system.

4- Renal failure, due to prolonged pressure on the intervening renal tissue.

Incidence:

Although congenital in origin, it usually presents at about 40 year.

Polycystic kidney

Clinical picture:

There are 3 clinical types according to the age of presentation.

1- Neonatal type, it presents with:

-Still birth or.

-Obstructed labour due to hugely enlarged kidneys.

2- Childhood type (3-7 years): It presents with:

-Renal rickets as polycystic kidney cannot preserve calcium.

-Renal failure.

3- Adult type (usually above 40 years): It presents with:

-Bilateral renal swellings.

-Pain: dull-aching (distension), colicky (obstruction) or dragging.

-Picture of complications.

Investigations:

1- Intravenous urography (IVU):

It shows elongation, attenuation and wide separation of calyces (by the cysts) giving spider-leg appearance.

Spider leg appearance

2- Ultrasonography. .

3- Renal angiography "to differentiate it from renal cystic tumors."

Treatment:

I- Conservative ttt:

1- Control of hypertension by salt restriction &antihypertensives but not diuretics.

2- Urinary antiseptics to guard against infection.

3- Dialysis if renal failure occurs.

II- Surgical treatment:

- Repeated puncture of superficial cysts to minimize pressure atrophy is obsolete because it is usually of no benefit and may introduce infection to the kidney.
- Renal transplantation:

It is the treatment of choice if renal failure occurred.

N.B. Nephrectomy is contraindicated before transplantation, as the disease is always bilateral.

Congenital solitary cyst

Aetiology:

1- Failure of communication as in polycystic kidney, but occurs in few numbers of nephrons.

2- Cystic dilatation of mesonephic remnants in the kidney.

Pathology:

Site: - It commonly occurs in the lower pole.

Composition:-It is filled with clear fluid.

Congenital solitary cyst

Complications:

1-Infection.

2-Haemorrhage: sudden severe pain.

3-Ureteric obstruction due to kink of the ureter.

4-Malignancy: in 5-10 % of all renal cysts, malignancy was detected.

Incidence:

Although congenital, it usually presents around the age of 40y, commonly in females.

Clinical picture:

I- Asymptomatic cases:

Most cases are discovered accidentally during routine investigations.

II- Symptomatic cases:

1- Pain: dull aching pain due to stretch of renal capsule.

2-Mass: tense cystic swelling in the hypochondrium. It has all characters of renal swellings.

3- Picture of complications.

Investigations:

1- IVU: It gives crescentic filling defect in the PC system usually pushing the ureter medially.

2- Ultrasonography: very helpful for diagnosis and aspiration.

3- Renal angiography: vessels are attenuated, and surround avascular area.

Differential diagnosis:

1- From other renal cysts.

.1- Renal abscess. 2- dermoid cyst. 3- Hydatid cyst. 4- Malignancy.

2-From Hypernephroma by renal angiography, C T scan, and aspiration.

3- From gastric & biliary dyspepsia.

Treatment:

I- Benign cysts: are treated with:

1- Aspiration and injection of sclerosant material e.g. 95% alcohol.

2- Enucleation. 3-Partial nephrectomy.

4- *Kirwin's operation:* -Deroofing of the cyst and marsipulizing it into fascia of Gerota. Painting of the floor of the cyst with carbolic acid may be added.

II- Malignant cysts:

They are treated with nephrectomy. Malignancy is suspected when:

- Red aspiration was withdrawn.
- Residual mass was left after aspiration.
- Rapid refilling after aspiration.
- Revealing malignant cells in cytology.

Aberrant & accessory renal arteries

Both aberrant and accessory arteries may compress over the ureter , or more commonly, they accentuate hydronephrosis for another cause.

Stewart nephroplication

Incidence:

-They are common in ♀.

-More on the Lt. side & bilateral in 25% of cases.

Treatment:

1- Division of the abnormal artery is not advised, as it may lead to ischaemia or infarction.

2-*Stewart nephroplication*plicating the upper & lower poles of the kidney with sutures to make the abnormal vessel cross over the wide pelvis rather than the narrow pelviureteric junction.

II- Trauma

1- Renal injury

Aetiology:

I- Predisposing factors:

1- Enlarged kidney, it becomes more exposed.

2- Diseased kidney, it becomes more fragile.

II- Precipitating trauma:

1- Blunt trauma (closed trauma).

a) Direct: blow to the lion.

b) Indirect: acceleration deceleration injuries e.g. fall from a height.

2- Penetrating (open) trauma: stab, & firearm injury.

3- Spontaneous rupture: due to minor unnoticed trauma.

Pathology:

Types

1- Extraperitoneal injury: (more common).

- It is more common in adults due to well-developed perinephric fat.

2- Intraperitoneal injury: less common.

- The post. peritoneum ruptures and blood enters the peritoneal cavity.
- It is more common in children due to underdeveloped perinephric fat.

Extent (degree):

1- Subcapsularhaematoma.

2- Small tears not communicating with the pelvicalyceal system.

3- Large tear communicating with the pelvicalyceal system.

4- Amputation of one pole.

5- Pulping of the kidney or shuttered kidney.

6- Avulsion of the renal pedicle.

Extent of injury

Complications:

I- General:

1- Shock. Haemorrhagic or neurogenic.

2- Haematuria (the commonest): It is absent in about 5% of cases due to:

a) Haematoma or tears not communicating with pelvicalyceal system.

b) Complete pulping of the kidney.

c) Avulsion of the renal pedicle.

d) Obstruction of the ureter by blood clots.

c) Injury to atrophied or non-functioning kidney.

3- Anuria due to:

a) Severe shock.

b) Obstruction of both ureters by blood clots.

c) Obstruction of one ureter while the other kidney is absent or non-functioning.

II- Local:

1- Collection of blood & urine around the kidney causing pseudo-hydronephrosis.

2- Infections: perinephric abscess. 2ry hage may occur

3- Ureteric obstruction by blood clot or later, by fibrosis causing hydronephrosis.

4- Increased mobility of the kidney.

5- Hypertension due to renal ischaemia.

Clinical picture:

I- History of trauma: (usually severe).

II- General exam:

1- Shock, neurogenic or haemorragic.

2- Haematuria, in 95% of cases.

3- Anuria, in severe cases.

III- Local exam:

1- Marked tenderness and rigidity in the hypochondrium& loin.

2- Swelling in the loin due to pseudohaemato- hydronephrosis.

3- Shifting dullness in cases of internal haemorrhage.

Investigations:

1- Abd U/S.: It can detect injury, its type & extent.

2- Plain X ray: It is suggestive, but, not diagnostic. It may show:

a) Fracture lower ribs.

b) Scoliosis of the vertebrae towards the injured side.

c) Blurring of the psoas shadow by the perinephrichaematoma.

3- I V U (Intravenous urography):

a) The injured kidney may not secrete the dye.

b) If it secreted dye, it may show:

- Extravasation of the dye from the pelvicalyceal system.
- Pooling of the dye into the kidney substance.

- Displacement of the kidney by haematoma.

4- CT, MRI: They are the most ideal investigation for haemodynamically stable patients, also it may reveal other organ injury.

5- Renal angiography with possible gel foam embolization.

6- Cystoscopy to detect the side of haematuria in bilateral cases with persistanthaematuria.

Treatment:

I- Conservative treatment.

It is successful in 80% of cases. It consists of:

1- Correction of shock: I.V fluids, bl.transfusion & morphia.

2- Antibiotics to guard against 2ry infection.

3- Catheterization to observe haematuria

4- Observation: -General: pulse, BP, urine output &haematuria by successive test tube test

-Local: Loin for swelling & rigidity.

-Laboratory & imaging: follow up CBC and abdominal U/S are ordered daily

-If he is stable, conservative measures are continued for at least 15 days in hospital for the fear of secondary haemorrhage.If the patient deteriorates, urgent operation is indicated.

II Angiographic embolization:

The treatment of choice for controlling renal haemorrhage when other indications for immediate surgical exploration do not exist

IIISurgical treatment

Indications:

1) Open injury.

2) Internal haemorrhage (Intraperitoneal).

3) Associated injury to other organs.

4) Failure of conservative treatment.

5) Late indication is to evacuate haematoma or drain perinephric abscess.

Procedure:

Anterior transperitoneal approach (laparotomy):

1- To inspect for other organs & internal haemorrhage.

2- To deal with the vessels first.

Technique:

It depends on the type of injury:

-Tear→ suture.

-Destruction or amputation of one pole→ partial nephrectomy.

-Pulped kidney or avulsion of the renal pedicle →nephrectomy.

2- Mobile Kidney

(Nephrpotosis)

Definition:

Hypermobile kidney that moves more than 2 inches during respiration. Normally, the kidney moves downward 1-2 inches during inspiration.

Aetiology:

I- Constitutional factors (متعلقة بالبنية):

1-Tall thin individual with shallow paravertebral gutter.

2-Weak anterior fascia ofGerotaand posterior Zuekercandle's fascia.

3-Long renal vessels.

4-Floating kidney, a rare anomaly where the kidney is completely covered with peritoneum and has a mesentery, which provides it a wide range of movement.

II Precipitating factors:

1- Trauma to the kidney.

2- Increased weight of the kidney e.g. hydronephrosis.

3- Sudden loss of perinephric fat as in regimen & starvation.

4- Sudden drop of intra-abdominal pressure as after labour or removal of huge abd. swelling.

Pathology:

It is commonly bilateral, but if unilateral, the Rt. side is more commonly affected.

Degrees: Normally, both kidneys are not felt except the lower pole of the right kidney, which can be felt, with difficulty in thin individuals. Hypermobility of the kidney has four degrees:

1st degree, the lower pole of the kidney is felt easily.

2nd degree.the upper pole is felt .

3rd degree, the kidney can be pushed out of the loin and back to it.

4th degree, (floating kidney) when the kidney can cross the midline of the abdomen.

Complications:

1- Obstruction of the ureter due to kink during descent leading to hydronephrosis.

2- Infection due to stasis.

3- Stone formation.

4- *Dietel's crises* due to acute kink of the ureter and renal pedicle causing severe abdominal pain with repeated vomiting and oliguria. It is precipitated by exercise and relieved by rest with passage of large amount of blood tinged diluted urine.

5- Neurosis: It was suggested due to traction on the sympathetic trunk.

Incidence:

It is more common in neurotic females.

Clinical picture:

I- Asymptomatic cases (20 % of cases).

II- Symptomatic cases:

A) Urinary manifestations:

1) Pain:

Character: dull aching. *Intensity*: mild to moderate.

Site:hypochondrium . *Referred to*: renal angle.

Increased by: exercise. *Decreased by*: rest.

2) Mass: One or both of the kidneys are felt with variable degree of mobility, but it can be always pushed back to the loin.

3) Picture of complications:

B] Gastrointestinal manifestations:

1- Traction on duodenum leads to epigastric pain & vomiting.

2- Traction of the colon leads to flatulence & constipation.

3- Traction on the CBD leads to biliary colic & jaundice.

Investigations:

1- Abdominal U/S: It shows the kidney in abnormal position.

2- IVU: it is taken with the patient erect with full inspiration & in Trendlenberg's position in full expiration. It shows: 1- The difference between the two positions is more than 2 inches.

2- The ureter is of average length but tortuous.

3- Aortography:It shows that the renal artery arises from its normal level but dragged down by ptosed kidney.

Treatment:

I- Asymptomatic cases:

No treatment & do not tell the patient about.

II- Symptomatic cases:

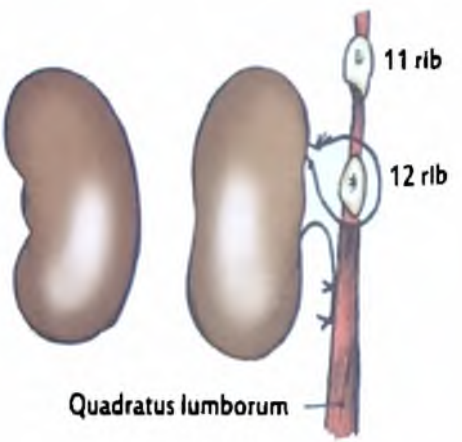
A- Mild symptoms without complications.

1- Reassurance.

2- Abdominal exercise to strengthen the muscles.

3- Abdominal corset.

4- Fatty diet to increase the perinephric fat.



El Katib's operation

B- Severe symptoms or complications = surgery.

1- *Elkatib's operation.*

-N shaped incision in the posterior capsule of the kidney and raising the two triangular flaps

-The upper flap is turn around the last rib and sutured to itself & the lower flap is sutured to quadratuslumborum muscle.

2- *Thompson walker's operation* simple sutures of the kidney capsule to quadratuslumborum muscle as the kidney has a high tendency for adhesions.

III- Inflammation

1- Acute pyelonephritis

Aetiology:

I- Causative organism.

E. coli (the commonest), proteus vulgaris & pseudomonas. Strept.faecalis& staph aureus.

II- Route of spread:

1- Ascending infection:

-The commonest route especially in females (short, wide urethera).

-It commonly follows instrumentation.

2- Haematogenous spread.

3- Lymphatic spread: from the colon.

III- Predisposing factors:

1- General factors: D.M. & debility.

2- Local factors: - Stasis -Stone or F.B -Excess coitus (Honeymoon pyelitis).

-Instrumentation or catheterization (catheter fever).

- Vesicoureteral reflux

Pathology:

There is diffuse oedema& congestion of the pelvicalyceal system & renal parenchyma.

Complications:

1- Chronicity: Ch. Pyelonephritis.

2- Toxaemia, septicemia, &pyaemia.

3- Primary pyonephrosis.

4- Perinephric abscess.

Incidence:

They are more common in females especially during pregnancy.

Clinical picture:

I- General:

-Fever (with rigor), headache, anorexia, & malaise.

II- Local.

-Pain in the loin &hypochondrium.

-Marked tenderness in renal angles.

-Rigidity in the hypochondrium.

-Pyuria: passage of turbid urine.

Investigations:

- Urine analysis to detect pus cells. If more than 100 Pus cells/HPF, Culture& sensitivity is performed to know the organism & suitable antibiotic.

Differential diagnosis:

From other causes of fever with rigor e.g. cholangitis, breast abscess, & malaria.

Treatment:

1- Broad spectrum antibiotics, which may be changed after culture & sensitivity.

2- There are two schools about induction of diuresis. The first advises plenty of fluids with diuretics to wash out infection and prevent stasis .Other school advises to keep the urine concentrated to obtain high concentration of antibiotics in urine.

3- Change the pH of urine.

-In acidic PH e.g. E.coli. alkalinization of urine can be obtained by use of Na bicarbonate .

-In alkaline PH e.g. staph &proteus (urea splitting organisms), acidification of urine is obtained by use of ascorbic acid (vitamin C).

4- Treatment of the predisposing factor.

2- Pyonephrosis

Definition:

Septic dilatation of pelvicalyceal system due to obstruction & infection. Lastly, the kidney is transformed into a bag of pus.

Pathology:

Types

1- Primary pyonephrosis (more common):

-It is due to coincidental infection & obstruction.

-It does not reach big size due to marked fibrosis of the kidney &perinephric tissue.

-The pelvis is not dilated and thick walled.

-The prognosis is bad.

2- Secondary hydronephrosis:

-It is due to infection of hydronephrosis.

-It is usually of huge size.

-The pelvis is dilated and thin walled.

-The kidney is filled with infected urine.

When obstruction is relieved, infection usually subsides and renal function improves

Complications:

1- Toxaemia, septicemia &pyaemia.

2- Descending infection.

3- Stone formation.

4- Renal failure if bilateral.

Clinical picture:

I- General:

Fever, headache, anorexia & malaise.

II- Local:

1- Mass: especially in 2ry pyonephrosis where it appears before inflammatory manifestations.

2- Pain: in the loin &hypochondrium.

3- Marked tenderness in the renal angle.

4- Rigidity in the hypochondrium.

5- Pyuria: It may be absent in closed (2ry) hydronephrosis .It is usually intermittent.

6- PR. Exam.: to detect SEP as a cause.

Investigations:

1- Urine analysis, for pus cells. Culture & sensitivity are important.

2- Kidney function tests.

3- U/S.

4- IVU: It detects type of pyonephrosis, level of obstruction & function of both kidneys.

Treatment:

I- **Unilateral cases** (with the other kidney functioning well).

1- If the diseased kidney is still functioning, treat the cause if possible e.g. stricture or stone.

2- If the diseased kidney is none functioning, nephrectomy is indicated.

II- **Bilateral cases** (or the other kidney is nonfunctioning).

Nephrostomy is performed (bilateral in bilateral cases).

1- If function improves, then treat the cause on the more hopeful side.

2- If the function does not improve, then dialysis until renal transplantation is available.

Genitourinary tuberculosis

Aetiology:

I- Causative organism: Mycobacterium tuberculosis, human type (75% of cases).

II- Route of infection:

1- Almost always haematogenous from T.B focus (may be T.B. mediastinal or mesenteric LN)

2- May be ascending infection from T.B prostatitis, seminal vesiculitis or cystitis.

III- Predisposing factors: Low resistance of the patient.

Pathology:

Renal tuberculosis is bilateral in 40% of cases.

1- Renal tuberculosis:

1) T.B. bacilluria: Infection of the kidney with passage of TB. Bacilli in urine with no gross pathology in the kidney.

2) Caseo-cavernous type: The T.B. follicles in the cortex coalesce together to form cavities in the kidney (cold abscesses).

3) Ulcero- cavernous T.B: Infection spreads into the medulla, reaches renal papillae of pyramids to form caseating lesions that bursts into calyces to form ulcers and finally, infection reaches the pelvis.

2- Ureters:

The ureter becomes thickened, fibrosed& shortened.

3- Bladder.

It is thickened, fibrosed& contracted (Thimble (كشتبان) bladder).

4- Genital organs.

Prostate, seminal vesicles & epididymis may be affected.

Complications:

I- Kidney:

1- TB. Hydronephrosis. 2- TB. pyonephrosis.

3- TB. Pseudo calculi: calcified TB. lesions in the kidney.

4- TB. autonephrctomy : due to destructed calcified kidney .

5- TB. perinephric abscess. 6- Miliary TB.

7- Renal failure 8- Haematuria.

II- Ureter: Stricture ureter.

III- Bladder: 1- Haematuria 2- Contracted bladder.

IV- Genital organs: Infertility.

Incidence:

It is more common in Young adults (20 - 40 y).female: male = 2: 1.

Clinical picture:

General:

TB toxaemia: Loss of weight, night fever & sweats.

Local:

1- *Frequency*: the earliest manifestation due to:

a) Polyuria: early due to congestion of the kidney, late due to failure of concentration of urine

.b) Irritation of urinary bladder by the caseous material.

c) Actual TB. cystitis or secondary infection.

d) Late due to contracted bladder.

2- *Pain*: which may be:

a) Dull aching pain in the loin (congestion of the kidney).

b) Colicky pain due to passage of caseous material.

c) Suprapubic pain referred to the tip of penis due to cystitis.

3- *Pyuria*: Passage of turbid urine.

4- *Haematuria*: - It is usually a late symptoms.

- It may be terminal (cystitis) or total (renal).

5- *Mass*: TB hydronephrosis, pyonephrosis or compensatory hypertrophy of the other kidney.

Investigations:

1- Urine analysis.

It shows low specific gravity, acidic pH, few pus cells, but no organisms. This *sterile acidic pyuria* is highly suggestive of urinary tuberculosis.

2- Bacteriological exam:specimens of early-morning urine

a) ZiehlNeelsenstain of centrifuged deposits (70%).

b) Guinea-pig inoculation (95%).

c) Culture on Lowenstein Jensen media (98%).

3- Kidney function tests.

4- Plain X-ray.

It may show pseudocalculi or calcified kidney.

5- Ultrasound. It detects hydronephrosis&pyonephrosis.

6- IVU .Early in ulcercavernous T.B, it shows moth (عثة) eaten appearance of the calyces.

late: It may show complication e.g. hydro or pyonephrosis, or contracted bladder .

7- Cystoscopy: It shows:

1-T.B. lesions in the bladder.

2-*Golf-hole appearance* of the ureteric orifice due to traction by the shortened ureter

8- Tuberculin test, ESR & chest X-ray to prove T.B aetiology.

Golf hole appearance

Treatment:

I- Medical treatment: for all cases.

1- Sanatorium admission.

2- Diet: good diet, vitamins & mineral.

3- Antituberculous drugs e.g. Rifampicin, INH, streptomycin, &pyrzinamide.

II- Surgical treatment.

Indications

1) Failure of medical treatment.

2) Presence of stricture or obstruction.

3) Development of complications e.g. pyonephrosis or autonephrctomy.

Operations

1- Renal cavernotomy:

-For caseating cavities not communicating with the pelvicalyceal system.

-The cavity (abscess) is aspirated first with wide bore needle, then the cavity is deroofed and the caseous material scrabed out of the cavity until the granulation tissue appears.

2- Nephro-uretrectomy:

For damaged nonfunctioning kidney, the ureter should be excised as low as possible (at the pelvic brim), not to leave a T.B focus.

3- Ileo-cystoplasty:

-For contracted bladder.

-Increasing the capacity of the bladder by anastomosing it to an isolated loop of the intestine with intact blood supply.

Ileo-cystoplasty

4- Renal carbuncle

Aetiology:

Causative organism: Staphylococcus aureus.

Route of spread:haematogenous, usually from cutaneous lesion e.g. furuncle, or Whitlow

Pathology:

Site: In the cortex under the capsule of the kidney.

-The lesion is an abscess with marked destruction of renal parenchyma. The cavity is filled with necrotic material with dense perinephric adhesion.

Complications:

1- Perinephric abscess, if it bursts into perinephric space.

2- Pyelonephritis if it bursts into a calyx.

Clinical picture:

General: constitution manifestations.

Local: tender ill-defined renal swelling.

Differential diagnosis:

From renal tumours (e.g. hypernephroma).

Treatment:

I- Medical ttt:

Antibiotics, analgesics & hot fomentation.

II- Surgery:

1- Drainage. by incision of the carbuncle, and removal of all necrotic materials .

2- Nephrectomy: if most of the kidney is destroyed.

Perinephric abscess

Aetiology:

I- *Causative organisms*: staphylococci, streptococci &E.coli.

II- *Route of infection:*

1- Haematogenous route (1ry perinephric abscess): rare, and from distant focus e.g. boil.

2- Secondary perinephric abscess:

- Extension from renal infection.
- Extension from nearby infection e.g. retrocaecal appendicitis, cholecystitis, pleura or spine

Pathology:

Site: Pus accumulates between the kidney and perinephric sheath.

Clinical picture:

I-General: hectic fever (with rigor), headache, anorexia & malaise.

II- Local:

-Pain with marked tenderness, hotness & rigidity in the loin.

-Loss of normal concavity (flattening) of the loin (fluctuation is very late).

-The urine is usually clear. If there is pyuria, this points to a renal cause.

Investigations:

1- Ultrasonography, very valuable.

2- Plain X ray & screening may show:

1) Blurring of psoas shadow by soft tissue shadow.

2) Scoliosis of vertebrae towards the diseased side.

3) Raised, fixed cupola of diaphragm.

3-IVU :In addition, it shows *Mathe's sign* i.e. No difference in the kidney levels in erect & supine position(loss of normal mobility of the kidney) due to dense perinephric adhesions.

Treatment:

I- Medical ttt:- Antibiotics analgesics, and hot fomentation.

II- Surgical ttt: → *Do not wait for fluctuation*

Drainage through lumber incision.

III- Treatment of the cause (source of infection) insecondary type.

IV- Renal stones(Lithiasis الحصى)

Definition:

Stones (calculi) are crystals laid down on organic matrix.

Aetiology:

I- Predisposing factors: Factors favoring stone formation can be classified into:

A- Extrinsic factors:

1- *Hot weather*: It causes excessive sweating and concentrated urine.

2- *Water supply*: Hard water, rich in calcium salts predisposes for stone formation.

3- *Dietary factors*:

1- Purine rich diet may precipitate urate stones (meat, liver, sardine and tuna).

2- Oxalate rich diet such as Mango, strawberry causeshyperoxaluria and oxalate stones.

3- Vitamin A deficiency: epithelial damage that gives rough surface for stone formation.

B- Intrinsic factors.

1- General factors:

[1] *Hypercalciurea*: as in hyperparathyroidism, sarcoidosis, & prolonged recumbency.

[2] *Hyperoxaluria* either primary hyperoxaluria or secondary to stagnant loop syndrome.

[3] Cystinuria: a congenital disease due to defect in cystine tubular reabsroption.

2- Local factors:-

[1] Obstruction: Stasis gives time for crystallization & predisposes for infection.

[2] Infection: infection is a good former for stone, as it provides:

1) Good nucleus: from tissue debris & bacterial colonies.

2) Good climate, as it changes PH of urine & colloid crystalloid ratio.

3) Good time: due to stasis.

4) Good soil: through ulcerations that provide rough surface for crystallization.

5) Good manures: albumin and fibrin of the exudate glue the constituents together.

[3] Change of urine composition:

1) Change of colloid-crystalloid ratio: Colloids (mucin&urochrome) maintain crystalloids in solution state. Disturbance of this ratio favour precipitation of crystalloids

2) Change of pH of urine: Alkaline PH favors formation of phosphate stones. Acidic pH is suitable for formation of other types.

Mode of formation of stone:

I-.Randall's theory:

-Crystals are deposited over ulcers at the tip of renal papillae (Randall's plaque). (may be due to Vit, A deficiency). When stones enlarge enough, they fall in the PC. system.

- This theory did not explain stones formed inside the renal parenchyma.

II- Carr's theory "Lymphatic pouching theory":

- It states that crystals are deposited inside parenchymal lymphatics that enlarge and pouch inside the PC. system. When they rupture, stones fall into the pelvicalyceal system.

- This theory explains the rare intrarenal stone.

Pathology:

Types of renal stone:

	Oxalate stone	Phosphate stone	Uric acid &urate stone	Cystine stone
Number	Usually single	Single or multiple	Usually multiple	Multiple
Size	Moderate	Large or staghorn	Small	Small
Shape	Irregular	Oval	Oval or faceted	Oval
Surface	Spiky	Smooth	Smooth	Smooth
Composition	Ca oxalate	Caphosph. or triple phosphate	Uric acid pure or	Cystine (sulphur containing AA.)
Colour	Dark (due to bl.)	Dirty white	Yellow to brown	Yellow light green
Consistency	Hard	Chalky, friable	Hard	Soft on light greasy (bee wax)
Cut section	Amorphous	Laminated	Amorphous	Amorphous
X ray	Radio opaque	Radio opaque	uric acid stone is translucent urate stone are radio-opaque	Radio opaque due to its sulphur content

Complications:

1] Migration causing colic.
2] Obstruction, hydronephrosis.
3] Infection.
4] Ulceration &haematuria.
5] Renal failure in bilateral cases.
6] Malignancy in long standing stone.

Incidence:

- It has a familial tendency e.g. familial hyper-oxaluria.
- It is prevalent in curtain districts e.g. districts using hard water.
- It affects male > females, more common in middle age.

Staghorn stone

Clinical picture:

I- Asymptomatic (silent) cases.

Cases may be discovered accidentally during routine investigations.

II- Symptomatic cases.

1- Pain: there are two types of pain.

a) *Fixed renal pain.*

Character, dull aching. *Intensity*: mild to moderate.
Site: loin. *Referred to*:hypochondrium.
Increased with: exercise. *Relieved by*: rest.

b) *Ureteric colic*: It occurs if a stone obstructs the pelviureteric junction.

Character, spasmodic. *Intensity*, severe.
Site: loin. *Referred to*: groin & scrotum or labia.
Increased with movement *relieved by*: antispasmodics

N.B. renal pain may be felt in the contralateral side "crossed renal pain".

III- Cases present with complications e.g. obstruction, infection or hematuria.:

Investigations:

I- Laboratory investigations.

1- Urine analysis for crystals, pus cells &pH.
2- Kidney function test.

II- Imaging studies:

1- U/S: Detection of stone and more importantly backpresure changes of the PC system.
2- Plain X ray: More than 95% of urinary stones are radio opaque "DD from radio-opaque shadow in the hypochondrium"

3-.Intravenous urography (IVU). It is mandatory to:

- Detect radiotranslucent stone " filling defects"
- Detect backpressure changes.
- Detect distal obstruction or predisposing factors.
- Detect the function of both kidneys.

Technique:

-1-2 amp of urographin (20-70 ml) are given IV,then serial X ray films are taken after 5, 10, 15 & 45 minutes and after voiding of urine (postvoiding film).

-The first film(s) for nephrogram. Zone of pressure may be created around the abdomen of the patient using two tennis balls and a binder to retain the dye in the kidney for somewhile.

-The film taken after 45 min is called descending cystogram, which normally shows the bladder, distended with the dye after evacuation of both ureters.

-Post voiding film is taken to detect residual urine after evacuation and ensure complete evacuation of the bladder.

Disavantages:

a)Hypersensitivity reaction to the contrast

b)Nephrotoxicity of the contrast IVU shouldn't be used when S.creatinine is > 2.5 mg%.

c)It is time consuming in emergency conditions e.g. trauma.

d)Kidney may not secrete the dye because of malfunction or spasm of renal artery due to pain

4- Three dimensions CT (Spiral CT): It is the most preferred imaging technique in emergency

5- Magnetic resonance urography (MRU): It is the preferred method in badly functioning kidneys

6- Radio isotope renal scan: to evaluate differential function of both kidneys.

III- Endoscopies.

1- *Cystoscopy* to detect predisposing factors e.g. SEP, cancer bladder, and ureterocele.

2- *Chromocystography*:

Injection of 4ml. 0.4% indigo-carmine solution I.V. It normally appears after 4minutes at the ureteric orifice. Delayed appearance for 5-10 minutes means mild to moderate impairing of function. Delayed excretion for 15 minutes means poor function.

3- *Uretroscopy&pyeloscopy*:

1- To visualize stones and may extract it.

2- To obtain ascending pyelography in poor renal function.

Differential diagnosis:

From other radio opaque shadows in the hypochondrium e.g. gall bladder stone, foreign body in the intestines & calcified LNs.

Treatment:

I- Conservative ttt:

- *Indications:*

1- Small stone < 1cm.
2- Smooth stone .
3- Sound kidney.
4- No Stricture below.
5- No Sepsis.
6- No Sequel e.g.hydronephosis.

- Protocol:

A- During the attack of renal colic: we give:

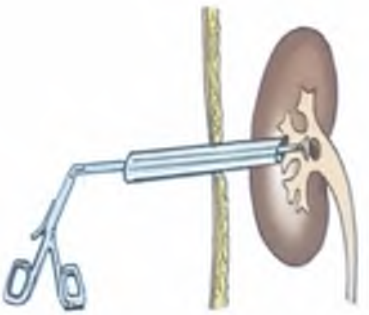
1- Anti spasmodics e.g. buscopan or glucolynamine.
2- Analgesics, up to morphia.
3- Antibiotics to "guard against infection".

In resistant cases:

1- Epidural anaesthesia.
2- Paravertebral sympathetic block.
3- Ureteric catheterization to release the obstruction.

B- Inbetween the attacks:

1- Dilution of urine by nigh water intake (3 liters /day)
2- Diuretics (interrupted course).
3- Dieting. the diet must be devoid of crystals..
4- Antiseptics.
5- Antispasmodics e.g. halphabarol.
6- Analgesics when needed.



Percutaneous nephrolithotomy

N.B.Alkalization of urine may help dissolution of urates&cystine stones. Pencillamine (chelating agents) may be used to avoid stone formation in cystinuria...

II- Extracorporeal shock wave lithotripsy (ESWL):

. Its principle is to expose the stone to shock waves created by electric explosion to pulverize the stone into small fragments that will pass spontaneously in urine within 1 week.

Advantages:

It is non-invasive method usually without anaesthesia

Contra-indications

1-Large stone (> 2 cms) or stag horn stone
2- very hard stones
3- Bleeding tendency
4- Morbid obesity (relative)

Complications

1- Renal colic during passage of fragments
2- Haematuria
3- Hypertension
4- subcapsularhaematoma.
5- Stien-strass (stones street) fragments accumulate over each other in the ureter obstructing it

III- Percutaneous nephrolithotomy (PCN):

- Under local anaesthesia, nephrostomy is done under IVU screen.
-Nephroscope is introduced into renal pelvis.
-Stone is extracted if small. If the stone is large, it could be crushed or pulverized by means of ultrasonic lithotripsy (USL) or electrohydrolic lithotripsy (EHL) probes, and then fragments are washed out by irrigation suction.

Complications:

1- Bleeding
2- peri-renal extravastion
3- Injury to the other viscera (duodenum, colon, liver and spleen).

Contra-indictions:

1- Bleeding tendency
2- Pregnancy
3- renal anomalies

IV- Surgical treatment:

Indications: rarely indicated nowadays

1- Big stone >2cm.
2- Spiky stone.
3- Deteriorating kidney function.
4- Presence of distal stricture.
5- Repeated sepsis.
6- Presence of hydronephrosis.

Exposure(incision):

Lumber incision = *Morris incision*. The incision starts 1 inch below the renal angle and passes parallel to the last rib to a point 1 1/2 inch above &antenor to the anterior superior iliac spine. We cut:

1- Skin &Sc.tissue.
2- Ext. oblique aponeurosis.
3- Internal abd. oblique.
4- Transversusabdominis.
5- Latissmusdorsi.
6- Serratus posterior inferior.
7- Renal fascia &perinephric fat.

Techniques:

1-Pyelolithotomy:

Indications

1- Stone in pelvis.

2- Stone in calyces can be brought out through the pelvis.

3- Stone in the upper 1/3 of the ureter.

Pyelolithotomy

2- Nephrolithotomy:

Indications;

1- When the kidney can't be brought out of the wound e.g. recurrent cases & obese patients.

2- Intrarenal stone.

3- Intrarenal pelvis.

4- Vascular anomalies in front of the pelvis.

- Extraction of the stone through incision in the kidney.
- The incision may be done over *Brodel's line*, which is 1cm from the convex border of the kidney on the posterior surface. It is the least vascular plane.

Brodel's line

3- Pyelonephrolihtomy:

Removal of stone by double approach. When we open the pelvis and the stone cannot be brought out through the pyelotomy, we can introduce our finger to push the stone towards the surface and the kidney tissue is incised directly over it.

4- Nephrotomy (not done nowadays):

It was indicated for big or staghorn stone.

The renal pedicle is temporary compressed by an assistant or clamped by vascular clamp, and the kidney is incompletely bisected through incision in its convex border down to the calyces to reach & extract the stone, then mattress sutures resuture it again.

It is not advised, as it destroys renal tissue and may be complicated with 2ry hage.

5- Partial nephrectomy:

In multiple recurrent stones in on one pole of the kidney with marked hydrocalycosis.

6- Nephrectomy:

*Indications.*The other kidney should be functioning well.

1- Multiple recurrent stones with bad function.

2- Marked hydronephrosis or pyonephrosis.

3- Uncontrollable bleeding during stone extraction.

Management of problematic casess:

I- Bilateral renal stones:

The role is to operate on the more hopeful side first, except if there is pain or infection on the less hopeful side. It should be operated upon first because:

1) Pain indicates obstruction to urine flow and the renal function will deteriorate rapidly.

2) Infection should be dealt with first to improve the general condition of the patient.

II- Two level stones:

For example, stone kidney & stone ureter, always deal with the distal one first because Dealing with the upper stone while the other stone is still obstructing the urinary passage will lead to development of urinary fistula from the proximal incision.

III- Staghorn stone:

1- PCN is tried first to pulverize stone and extract fragments as complete as possible.

2- ESWL is done to deal with calcyceal fragments through successive sessions.

3-If this failed, pyelolithotomy is tried. If delivery is difficult, the stone is crushed into fragments, which could be extracted by Pyelonephrolihtomy.

4- Staghorn stone in old unfit patient with bad function may be managed conservatively.

Obstructive Uropathy

Hydronephrosis

Definition:

Aseptic dilatation of the PC.system due to intermittent or incomplete distal obstruction .

Aetiology:

I- Pelviureteric causes (unilateral or bilateral):

A- In the lumen:

Stone (calcular obstruction).

B-In the wall:

1- *Congenital*: Congenital ureteric stricture.

2- *Inflammatory*: - Bilharzial stricture (the commonest in Egypt).

- T.B structure.

3- *Neuromuscular*:pelvi ureteric achalasia.

4- *Neoplastic*: -Cancer bladder infiltrating ureteric orifices.

-Renal pelvis tumours obstructing the pelvis.

C- From outside:

-Aberrant renal artery.

-Paraureteric bladder diverticulum compressing on the lower ureter.

-Retroperitoneal fibrosis.

-Cancer cervix or gravid uterus compressing the ureter.

Renal hydronephrosis

II- Vesico - urethral causes (bilateral cases):

A-In the wall:

1- Congenital: Congenital bladder neck obstruction.

2- Inflammatory: - -Bladder neck obstruction.

- Stricture urethera.

3- Neuromuscular: Marion's disease.

4- Neoplastic:.-Senile enlargement of prostate (SEP) .

-Cancer bladder or cancer prostate .

Pelvic hydronephrosis

Pathology:

Types.

1- Renal hydronephrosis:

-It occurs when the pelvis is intra-renal.

-Dilatation of the calyces on the expense of the renal parenchyma.

-At first, there is loss of the normal cupping of the calyces, then there is clubbing then ballooning of the calyces.

-The prognosis is bad.

Pelvi-renal hydronephrosis

2- Pelvic hydronephrosis.

-It occurs when the pelvis is extrarenal.

-The pelvis dilates markedly and may form a spur between it & the ureter that acts as a valve increasing the obstruction.

Hydrocalycosis

-At first, the backpressure changes of the calyces are minimal, then the calyces dilate and this is called pelvi-renal hydronephrosis.

3- Hydrocalycosis:

-when obstruction involves the neck of a calyx.

Complications:

1- Stone formation due to stasis & infection.

2- Infection & stasis invites infection.

3- Large kidney more liable to trauma.

4- Increased mobility of the kidney.

5- Hypertension due to renal ischaemia.

6- Renal failure if bilateral.

Clinical picture:

I- Asymptomatic cases: Some cases are asymptomatic for a long time.

II- Symptomatic cases:-

1- *Pain*: 1- Colicky pain due to obstruction by stone (the cause).

2- Dull-aching pain in the loin due to distension.

2-*Mass*: which has all renal criteria (see later)?

III- Cases present with complications.

IV- PR exam.: can detect the cause e.g. SEP.

Investigations:

I- Laboratory:

1- Urines analysis: Crystals, RBCs, pus cells. 2- Renal function tests:

II- Imaging studies:

1-*Abd.U/S*: -It is very helpful to diagnosis type , cause & thickness of renal cortex.

2-*Plain Xray* (KUB) may show:

1) Enlarged soft renal shadow.

2) Stone pelvis, or ureter as an underlying cause or stone kidney as a complication.

3- *Intravenous urography* (IVU). It shows:

1) Function of both kidneys.

2) Type of hydronephrosis (renal, pelvic, pelvirenal or hydrocalycosis).

3) Degree of hydronephrotic changes e.g. flattening, clubbing, or ballooning of the calyces.

4) The underlying cause.

4-*Intravenous infusion urography*:

If the function of the affected kidney is poor, urographin is given by IV infusion drip to give a chance for the diseased kidney to secrete it sufficiently to be visualized in X ray films

5- *Ascending pyelography*:

After ureteric catheterization, the dye is injected to opacify the pelvis & ureter.

III-Renal isotope scan:

To detect the remaining renal tissues using radio-active hippuran or DMSA.

IV- Endoscopy:

1-*Cystoscopy &Uretroscopy*:

To diagnose the cause e.g. cancer bladder, stone ureter, and stricture.

2-*Chromocystoscopy:* to evaluate kidney function.

Differential diagnosis:

From other kidney swellings.

Treatment:

I- Unilateral cases (the other kidney is functiong):

1- If the kidney is still functiong, treat the cause.

2- If it is not functioning, do nephrectomy.

II- Bilateral cases (or the other kidney has a poor function).

Do nephrostomy (bilateral nephrostomy in bilateral cases) until the kidney function improves.

1) If the function improved, treat the causes on the more hopeful side.

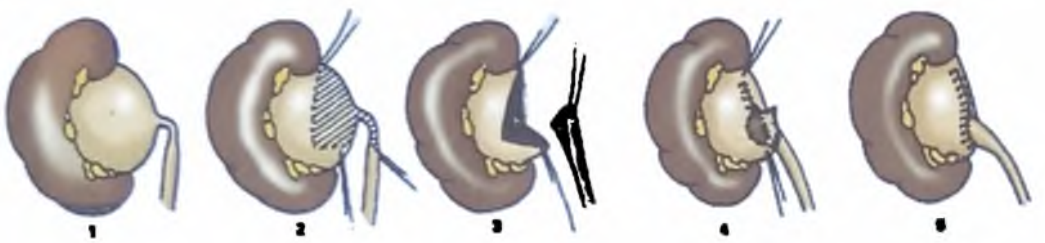
2) If the function did not improve maintain nephrostomy & prepare for renal transplantation.

Examples for treatment of the cause.

I- In pelviureteric stricture.

1)Andreson-Hyne's operation:

Resection of the redundant pelvis, stricture, and reconstruct a new wide pelvi-ureteric junction.



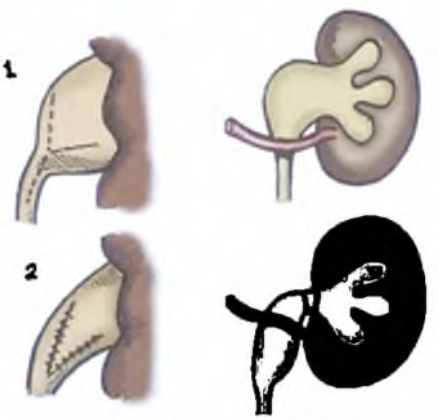
Andreson-Hyne's operation

2) Y-V pyeloplasty.

Y shaped incision of the pelviureteric junction which is closed in V manner to widen the stricture

II- Aberrant renal vessels:

Stewart nephroplication: plicating the upper & lower poles of the kidney with sutures to make the abnormal vessel cross over the wide pelvis rather than the narrow pelviureteric junction.



Y-V pyeloplasty **Stewart nephroplication**

V- Neoplasms

A- Benign tumours

Adenoma, haemangioma (rare).

B- Malignant tumours.

Tumours arising from renal parenchyma

1-Renal cell carcinoma (75%)

2-Wilm's tumour (10%)

3-Leiomyosarcoma, haemangiosarcoma (5%)

Tumours arising from renal pelvis (10%)

1-Transitional cell carcinoma.

2-Sq. cell carcinoma.

1- Renal cell carcinoma

= Hypernephroma = Grawitztumour

Aetiology:

I- Cell of origin:

It was thought that it arises from the suprarenal gland, but this belief is not accepted due to:

1- The tumour does not secrete adrenal hormones.

2- The tumour may arise from the lower pole.

3- The tumour may show tubular structure resembling renal tubules.

Now, it is believed that it arises from the proximal convoluted tubules.

II- Predisposing factors:

- *Age*: It occurs most commonly in the fourth and the fifth decade of life
- *Sex*: The male: female = 2: 1.
- *Race*: It is commoner in Eauropean and Americans than in Africans and Asians.
- *Genetics*: It runs in families. Loss of the short limb of chromosome 3
- It is more common in smokers, obese, hypertensive and people under oestrogen therapy.

III- Premalignant conditions.

- Renal adenomas.

Pathology:

Site:

It usually arises in one pole of the kidney usually the upper pole.-Bilateral in 1-2 %.

MAC:

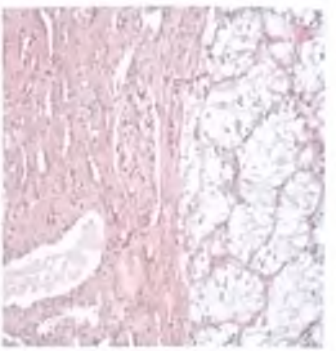
-The tumour is spherical, variable in size, golden yellowish in colour due to its high lipid content. The tumours are falsely capsulated by the surrounding tissue, but there are satellite lesions beyond the false capsule. The tumour infiltrates the renal pelvis early.

-The tumour is divided by multiple septae into numerous lobules.

MIC:

1-Clear cell adenocarcinoma: solid masses of rounded or polyhedral cells with dark stained nuclei and clear cytoplasm. The clear appearance of cells is due to dissolution of lipid content of cells during alcoholic preparation.

2- Dark cell type: the cells have dark granules (mitochondria). Sometimes, the two types of cells coexist.



Clear cell adenocarcinoma

Spread:

1-Direct:

Intrinsic: it invades the pelvis early causing haematuria.

Extrinsic: it infiltrates the perinephric tissues and later, to adjacent viscera.

2-Lymphatic spread:

To hilar lymph nodes, then to para-aortic nodes. Finally, thoracic duct and Virchow's LNs may be involved.

3-Blood spread (the commonest route).

Embolization: to the lung (canon ball metastasis), bone, and brain.

Permeation: hypernephroma has a tendency to invade the renal vein as solid columns of cells that may extend to IVC.

Staging: TNM staging

T1 = tumour 7 cm or more, limited to the kidney.

T2 = tumours more than 7 cmlimited to the kidney

T3 = tumour invading the veins, adrenal or perinephric fat but confined to fascia of Gerota

T4= tumour extending beyond fascia of Gerota.

N0 = No regional LNs involovement.

N1 = Single regional LN involovement.

N2 = more than one regional LN involovement

M0 = No evidence of distant metastasis

M1 = Distant metastasis

Complications:-

1- *Haematuria*: early & common.

2- *Hypertension*: due to renin-angiotensin system.

3- *Unexplained fever*.

4- *2ry varicocele*: due to obstruction of renal vein.

5- *Polycythaemia*: erythropoietin.

6- *Distant metastasis*.

7- *Paramalignant syndrome*: It may secrete parathormone like substance →hypercalcaemia.

Incidence:

- It is the commonest malignant renal tumours (75%).
- More common in males (♂: ♀ = 2: 1), more in the age of 50 - 70 years.

Clinical picture:

1-*Haematuria* (90% of case):

- The presenting symptom in most cases.
- It is painless, periodic, and profuse panhaematuria.

2- *Pain* (in 40% of cases):

a- Dull aching due to stretch of renal capsule.

b- Colicky due to passage of blood clots.

c- Severe boring due to infiltration of lumber nerves (rare & late).

3- *Mass*: (in 50% of case): irregular hard renal mass, early mobile, but later, it becomes fixed.

4- *Picture of complications*. e.g. hypertension, fever, weight loss ,& signs of metastasis .

Investigations:

1- Urine analysis, for RBCs, pus cells, malignant cells by cytology.

2- Kidney function tests.

3- Ultrasonography: can detect most tumours and detect cystic changes.

4- IVU: It may show elongation, distortion or amputation of a calyx.

5- CT scan & MRI: detect invasion of renal capsule, renal vein or regional LNs involvement.

6- Renal angiography: It shows abnormal vessels and high vascularity of the tumours.

7- Cystoscopy to detect the side of bleeding in cases presenting with haematuria.

8- Ureteric catheterization & ascending pyelography: if the function is poor.

9- Investigations to detect metastases: Plain X ray or CT of chest &abdomen bonescan.

Differential diagnosis:

I- From other causes of haematuria.

II- From other renal masses: see later.

Treatment:

I- Operable cases :

Radical nephrectomy: We remove:

-The kidney & adrenal.

-The perinephric fat &Zuekercandle's fascia.

-The regional lymph nodes.

-The renal pedicle & upper 1/4 of the ureter.

Abdominal approach is preferred because:

1-It allows ligation of the renal vascular pedicle first, so avoid dissemination of malignant cells during manipulation of the kidney, and enable removal of possible malignant thrombus from renal vein or even IVC.

2- It allows abdominal exploration and dealing with infiltrated viscera if present.

3- It allows removal of large tumours.

* Now laproscopic nephrectomy can be performed but only for T1 cases

* Nephrone saving surgery i.e. partial or subtotal nephrectomy indicated in bilateral cases or RCC in solitary functioning kidney

II Inoperable cases:

Radiotherapy, chemotherapy & immunotherapy (using cytokines e.g. interferon, & or interleukin –2).

Prognosis:

Operable cases have a 5-year cure rate of about 60% after nephrectomy.

2- Wilm'stumour

Nephroblastoma

Aetiology:

I- Cell of origin: It arises from embryonic nephrogenic tissue (multipotent mesodermal cells) remaining in the kidney.

Pathology:

Site:

-It arises from one pole of the kidney; usually the upper one.-It is bilateral in 5% of cases

MAC:

-Usually a huge swelling, pinkish or grayish white in color.

-The cut section shows areas of haemorrhage & necrosis.

MIC:

-The tumour contains two types of tissues:

-Epithelial element: in the form of abortive tubules & glomeruli.

-Connective tissue element: in the form of muscle fibres, cartilage and even bone.

*The degree of differentiation varies from favorable to unfavorable histology

Spread:

I- Direct: Invades the capsule and surrounding tissues, but later, it infiltrates renal pelvis.

II-Lymphatic spread (rare): to hilar& lumber LNs.

III-Blood spread to lung, bone & liver.

Staging: It is a post-nephrectomy staging.

Stage I: The tumour is confined to the kidney and was completely excised.

Stage II: The tumour extends beyond the kidney but was successfully completely excised.

Stage III: Residual into abdominal (non-haematogenous) tumour.

Stage IV: Distant haematogenous or lymphatic spread.

Stage V: Bilateral renal involvement.

Complications:

1- Destruction of renal tissue.
2- Weight loss.
3- Haematuria in 20 %.
4- Fever in 3%.
5- Hypertension in about 50% of cases.
6- Distant metastasis.

Incidence:

-It accounts for 80 % of genitourinary cancers (8 % of all malignant tumours) in children.

-The commonest age affected is the first 4 years. 80% of cases are between 1 - 5 years of age.

- Male : female = 1 :1..

Clinical picture:

I-*Mass*: in almost all cases, and usually is rapidly growing huge mass causing huge abdominal distension in emaciated infant "spindle shaped infant".

II- *Haematuria*: only in 20 % of cases.

III- *Pain*: in 20% of cases. It is dull-aching due to stretch of the capsule.

IV- *Manifestations of complications* e.g. hypertension, metastasis, & unexplained fever.

Investigations:

1- Urine analysis for haematuria..

2- Kidney function tests.

3- Plain X ray: marked enlargement of soft renal shadow.

4- IVU:

- Early displacement & attenuation of pelvicalyceal system.
- Late: No function of the affected side.

5- Ultrasonography, CT scan or MRI:

- They diagnose stage, assess operability, and follow up after management

Plain X ray

Differential diagnosis:

From other huge abdominal swellings in children:

1- Infantile polycystic kidney. 2- Multicystic kidney

3- neuroblastoma 4- lymphoma.

Treatment:

-Radical nephrectomy is done and according to the post nephrectomy staging, subsequent treatment varies as follows:

Stage I: Chemotherapy with actinomycin D & vincristine for 6 months.

Stage II: Chemotherapy with actinomycin D, vincristine &adriamycin for 15 months.

Stage III: Radiotherapy to the residual tumour, & chemotherapy as stage II.

Stage IV: or any stage with unfavorable histology:

-Radiotherapy +Chemotherapy but for 16 months.

Stage V: Radical nephrectomy on one side & partial nephrectomy on the other side or bilateral Nephrectomy + renal transplantation + radiotherapy & chemotherapy as before.

NB. Huge tuomours and irresectable tumours should have pre-operative chemotherapy then nephrectomy then post-operative chemotherapy

Prognosis:

The 5 years survival rate is 80% for infant below 1 year.

Renal pelvis tumour

I- Transitional cell carcinoma (TCC)

(Papillary carcinoma)

Aetiology:

I-Cell of orgin: Transitional epithelium of the renal pelvis.

II-Predisposing factors :

1- Tobacco smoking. 2- Phenacetine abuse. 3- Aniline dye intoxication.

III- Premalignant condition:

Villous papilloma (Grade 1 cancer).

Pathology:

MAC: Pedunclated mass with multiple fronds like projections with marked vascularity.

MIC: Transitional cell carcinoma.

Spread: Commonly by surface implantation on ureter & urinary bladder. Patients with TCC have 30% chance for developing the same lesions in the bladder.

Clinical picture:

The main constant presentation is profuse persistent haematuria.

If it caused obstruction, it will give rise to renal mass (hydronephrosis).

Investigations:

1- Urine analysis: For RBCs & cytology for malignant cells.

2- IVU: It may show filling defect in the renal pelvis.

3- Pyeloscopy: To visualize the tumour and to take a biopsy.

IVU in renal pelvic tumour

Differential diagnosis:

From radiolucent pelvic stone

Treatment:

Extended Nephro-uretrectomy with excision of a cuff from the bladder around ureteric orifice.

2- Squamous cell carcinoma.

Aetiology:

Predisposing factors: Long-standing stone in the pelvis →sq. metaplasia →sq.cell carcinoma.

Pathology:

MAC: nodular ulcerating mass invading the renal pelvis & parenchyma.

MIC: squamous cell carcinoma.

Spread: As before but surface implantation: does not occur.

Incidence:

More common in old males.

Clinical picture:

1- *Haematuria*: less marked.

2- *Mass*: if it caused obstruction.

3- *Pain*: dull aching with obstruction, colicky with passage of blood clots (clot colic).

The diagnosis is usually late, as it usually occurs on top of long standing stone and both the patient & his physician are accustomed to the attacks of pain &haematuria.

Differential diagnosis:

From radiolucent pelvic stone

Treatment:

Most cases are inoperable when first diagnosed, due to the late diagnosis.

Prognosis:

It has the worst prognosis among all renal tumours due to its late diagnosis & radio resistance

The Ureter

Anatomy of the ureter

-It is a tubular organ about 25 cm in length joining the renal pelvis and the urinary bladder.

-It descends on the posterior abdominal wall and pelvic floor crossing the following structures:

1- Muscle: psoas major muscle.

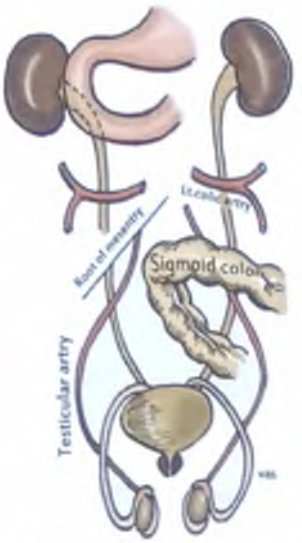
2- Nerve: genito femoral nerve.

3- Artery: Bifurcation of common iliac artery.

4- Joint: sacroiliac joint.

5- Bone: tip of ischial spine.

6- Fat: fat over pelvic floor (levator ani).



Ant. relations of the ureters

Both ureters have the same posterior relations, but they vary in their anterior relations as follow:

Rt ureter	Lt. ureter
- The 3rd part of the duodenum.	
- Rt. colic & iliocolic arteries.	- The Lt. colic artery.
- Root of the mesentry.	- the sigmoid mesocolon
- The Rt. gonadal vessels.	- The Lt gonadal vessels.
- Rt vas deferens in ♂ and broad lig. and uterine artery in ♀.	- Lt. vas deferens in ♂ and board lig. and uterine art. in♀.

The ureter has five constrictions.

1- At the pelviureteric junction.

2- Where it crosses the bifurcation of the common iliac artery.

3- Where it crosses the vas deferens in male or broad ligament in female (Juxta position).

4- Where it enters the wall of the bladder.

5- The ureteric orifice.

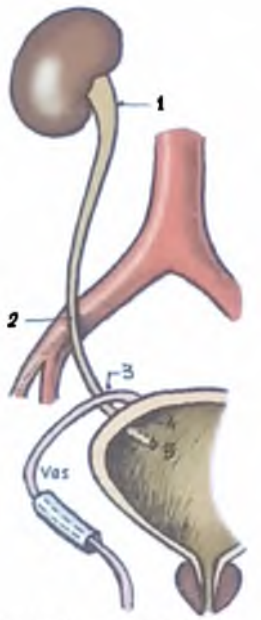
These sites are:

-The common sites for stone impaction.

-The common sites of inflammatory strictures.

-The common sites of injury during instrumental maneuvers.

Blood supply and lymphatic drainage of the ureter are segmental as it gains them from different regions through which it passes.



Ureteric constrictions

The ureter is surgically divided into three parts:

1- Upper 1/3: extending from its beginning to the iliac crest.

2- Middle 1/3: extend from the sacroiliac joint to the tip of the ischial spine.

3- Lower 1/3: from the ischial spine down to the ureteric orifice.

The ureter is identified during operation by the following data:

1- It is a tubular structure running longitudinally on the posterior abd. wall with longitudinally arranged bl. vessels over it.

2- It is attached to the back of peritoneum by losse areolar tissue.

3- An important landmark for the ureter is the point at which it crosses the bifurcation of the common iliac artery.

4- It shows periodic peristalsis.

In IVU. the ureter appears crossing .

1- The tip of transverse processes of lumbar vertebrae.

2- The sacro iliac joint.

3- The tip of ischial spine.

IVU

I- Congenital anomalies

1- Single ureter :

- It is associated with unilateral renal aplasia.

2- Double ureter :

-It is associated with supernumerary kidney or bifid pelvis.

3- Postcaval ureter :

- The right ureter passes behind the IVC and may be obstructed by it.

4- Ectopic ureteric orifice:

-It is associated with duplication of the ureter.

Double ureter

-Usually it is the upper member of a double ureter that opens ectopically lower.

-In males, the ectopic opening may be in the posterior urethra, seminal vesicles or ejaculatory ducts, but in females, the ectopic opening may be in the urethra, vagina or vulva.

-As the ectopic orifice in males is always proximal to the external urethral sphincter, so it is silent. In females, it leads to incontinence, as it lies distal to the sphincter.

Treatment:

In males: no treatment.

In females: transplantation of the ectopic ureter into the bladder.

5- Ureterocele:

Definition: Cystic dilatation of the distal end of the ureter projecting into the bladder.

Aetiology: due to congenital pinhole ureteric meatus leading to distension and ballooning of the intramural portion of the ureter, and may be with prolapse of its mucosa into the bladder.

Clinical picture:

1) Frequency due to - irritation of the bladder by prolapsed mucosa or Infection.

2) Picture of complications e.g. infection, & hydronephrosis.

Ureterocele

Investigations:

1) IVU: shows the characteristic *cobra head* appearance.

2) Cystoscopy.

Treatment: Cystoscopic diathermy incision is usually all that should be done.

II- Ureteric stones

Aetiology:

I- Source of stones:

1- Primary formed stones in the ureter (rare).

2- Migrating stone from the kidney (most cases).

II- Predisposing factors: For stone formation, see renal stone.

Pathology:

Sites: The stones are usually arrested at one of the normal constrictions (where?) and usually in the lower ones i.e. in the pelvic part of the ureter.

Types of stones. See renal stones.

Shape: Primary formed stones usually takes an elongated shape (date shaped).

Complications:

1- Migration (ureteric colic).

2- Obstruction hydroureter, & hydronephrosis.

3- Ulceration, causing haematuria.

4- Infection.

5- Renal failure, if bilateral.

6- Calcular anuria due to bilateral impacted stones or unilateral impacted stone in a single kidney.

Incidence:

As in renal stones.

Clinical picture:

I- Asymptomatic cases:

Minority of cases is asymptomatic and discovered during routine investigations.

II- Symptomatic cases:

1- Pain:

-Character: *colicky in migrating stone. *dull-aching in impacted stone.

-Intensity: moderate to severe.

-Site & reference:

* Upper 1/3 is felt in the loin & referred to the groin & scrotum in ♂ or labia majora in ♀.
* Middle 1/3 is felt in the iliac fossa (on the right side, it may simulate appendicitis).
* Lower 1/3 is felt in suprapubic region and is referred to the tip of penis.

-Increased by jarring. -Decreased by strong analgesic & antispasmodics.

-Associated with: nausea & vomiting.

2- Frequency: in a stone at the lower end of the ureter.

3- Picture of complications e.g. renal mass, fever, pyuria, haematuria & calcular anuria.

III- PR exam. to detect predisposing factors e.g. SEP or cancer bladder.

Investigations:

1- *Urine analysis*: for crystals, RBCs, pus cells, & ova.

2- *Kidney function tests.*

3- *Abdominal U/S*: To detect pressure changes on the kidney and less frequently the stone.

4- *Plain X-ray:*

95% of stones are radiopaque and can be detected along the course of the ureter.

5- *IVU*: It shows

- Radio translucent stones as filling defect.
- The predisposing factors e.g. SEP or stricture ureter.
- The complications e.g. hydroureter, or hydronephrosis.
- Function of both kidneys.

6- *Renal isotope scan*: to assess the function of both kidneys.

7- *Cystoscopy*, for:

- Detection of a stone in the intramural part of the ureter (peeping stone).
- Detection of predisposing factors e.g. SEP or cancer bladder.
- Detection of the side of bleeding in case of haematuria.
- Ureteric catheterization to: - do ascending pyelography to detect the site of stone.
  -drain infected urine above (relieve obstruction).
  -try stone extraction.

8- *Ureteropyeloscopy*: for visualization of the stone & trial of extraction.

Differential diagnosis:

1- Stone middle third ureter from acute appendicitis.

2-Stone lower end ureter from other causes of stone in high lateral position.

Treatment:

I- Conservative treatment:

The same indications & medications as renal stones.

II- Extracorporeal shock wave lithotripsy:

It is tried but usually unsuccessful as there is no room for shock waves.

III- Instrumental treatment:

1- Ureteric catheterization.

-Especially if the stone is obstructing, a catheter is tried to pass above the stone to relieve obstruction & drain urine and is left in place for 48 hours to relieve oedema & congestion. Meanwhile, injection of lubricants & antispasmodics is done to facilitate stone descent.

-When the catheter is removed, stone usually follows it within 48 hours.

2- Stone extraction:

-*Dormia basket* is passed above the stone, then opened and withdrawn with the stone.

- Ureteric injury may occur if the stone is large.

- Large stone may be disintegrated first by USL or EHL then extracted.

3- Endoscopic meatotomy:

When stone is impacted in the intramural part of the ureter.

N.B Instrumental treatment is of little value in Egypt due to prevalence of bilharziasis, which causes lower ureteric stricture.

IV - Surgical treatment (open removal).

1- Stone upper 1/3:

Through lumber or Morris incision, the stone is extracted by pyelolithotomy.

2- Stone middle 1/3:

-Through *Abernathy's incision*, which is done 2 inches above, and parallel to the lateral 1/2 of the inguinal ligament then it may be extended laterally with an upward curve.

-It is muscle cutting incision that cuts the two oblique muscles & the transversus abd. muscle

3- Stone in the lower 1/3: Through a midline suprapubic, incision from the umbilicus to the symphysis pubis the ureter is reached extraperitoneally.

Advantage of the suprapubic approach.

1- Both ureters can be explored though the same incision.

2- If stone is impacted in the intramural part of the bladder can be opened and stone can be extracted through meatotomy (transvesical ureterolithotomy).

3- The bladder can be operated upon in the same sitting if needed.

Management of problematic stones:

1) Double level stones: The distal stone is operated upon first to release distal obstruction.

2) Bilateral stones: The stone on the more functioning side is operated first, except if there is pain or infection on the less hopeful side, where it is operated upon first.

II-Ureteric strictures

Aetiology:

I- Congenital: rare and usually in the intramural part or pelviureteric junction.

II- Traumatic: after operative trauma, long standing stones or radiotherapy.

III- Inflammatory: Stricture or TB stricture.

IV- Neoplastic: 1ry carcinoma of the ureter is very rare

2ry carcinoma (more common) e.g. cancer Cervix, uterus, colon & rectum.

Bilharzial stricture of the ureter

Aetiology:

I- Causative organism:

S. haematobium & much less commonly, S. Mansoni.This is because the former is more slender and can pass easier through the anastomosis between rectal & vesical plexus.

II- Life cycle of Bilharziasis. (See before).

III-Pathogenesis:. Stricture occurs due to dense fibrosis around deposited bilharzial ova.

Pathology:

Site: It is common in:

- Site of normal anatomical narrowing especially the lower constrictions, e.g. intramural & juxtaposition strictures.
- Opposite the transverse process of L3 *(Makar stricture).* This is due to venous anastomosis between the colon & the Lt ureter.
- It is commonly bilateral, and if unilateral, it is more common on the Lt. side.

Complications:

1- Obstruction (stasis) leads to hydroureter & hydronephrosis.

2- Infection (sepsis) with recurrent pyelonephritis.

3- Stone formation, due to the previous two factors.

4- Renal failure if bilateral & neglected.

5- Vesico-ureteric reflux 2ry to intramural part involvement.

Incidence:

-It is common in Egypt due to prevalence of bilharziasis.

-It is more common in young adult male farmers.

Clinical picture:

I- History of contact with infected water.

II- Picture of B cystitis & BNO:

1- Pain: suprapubic, referred to the tip of the penis.

2- Frequency of micturition.

3- Terminal haematuria.

4- Difficulty of micturition when BNO occurs.

III- Picture of complication e.g. hydronephrosis pyelonephritis or renal failure.

Investigations:

1-Urine analysis: for RBCS, pus cells, crystals & B. ova.

2-Kidney function tests.

3- Abd. U/S. to detect hydroureter, & hydronephrosis.

4- Plain X ray may show: -calcified urinary bladder.
-calcified stricture ureter.
-stone as a complication.

5- IVU may show: -Hydroureter, & hydronephrosis.
-The level & length of stricture.
-The function of both kidneys.

Bilharzial strictures with bilateral hydroureter

6- Cystoscopy: It may show: -Bilharzial lesions of the bladder.
-Ureteric catheterization will arrest at the level of stricture.
-Ascending pyelography if the renal function is poor.

Treatment:

I- Medical treatment of bilharziasis antibilharzial drugs.

II- Instrumental treatment cystoscopic meatotomy if narrowing affects the ureteric orifice but usually the B stricture is relatively long stricture.

III Surgical ttt:

A- Stricture in the lower ureter one of the following operations

1- Operative dilatation: recurrence is the rule.

2- Transvesical excision advancement with nipple formation.

-It is indicated in the intramural or juxavesical stricture.

-The bladder is opened, and an incision is made around the ureteric orifice through the wall of the bladder. The ureter is pulled through the bladder wall and the strictured segment is excised. The healthy ureter above is sutured to the bladder with eversion of the ureter into the bladder to form a nipple in order to avoid reflux of urine.

3-Uretero vesical reimplantation:

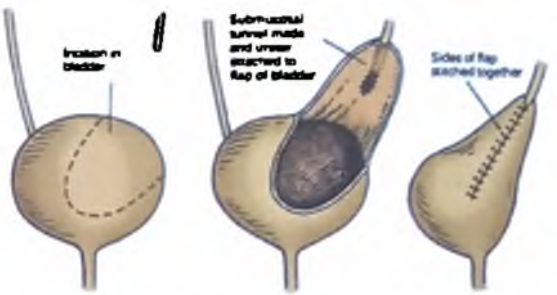
-It is also indicated in intramural or juxavesical stricture.

-The ureter is divided above the stricture and implanted into the bladder near its original orifice better through *a submucous tunnel* or nipple formation to avoid reflux of urine.

4- Boari's cystoureteroplasty:

-A rectangular flap from the bladder is fashioned into a tube and anastomosed to the ureter after excision of the stricture.

-The flap contracts with the bladder to prevent the reflux of urine into the ureter.



Boari's cystoureteroplasty

B)-Stricture of the upper two thirds.

A-*Short stricture* (annular stricture).

1-Excision & end to end anastomosis.

2-Ureteroplasty, longitudinal incision closed transversely.

B-*Long stricture.*

1-Bridging the gap after excision of the stricture with ileal or appendicular flap.

2-*Davis intubated ureterostomy* (especially for recurrent cases)

The stricture is opened longitudinally and is left to heal over a T tube.

Treatment of the problematic cases

If the renal function is poor.

-If the other kidney is healthy nephrectomy is indicated .

-If the other kidney is bad function, nephrostomy is performed until renal function is improved:

*If improved, treat the stricture on the more hopeful side.

*If not improve, treat the case as renal failure and prepare for renal transplantation.

The Urinary bladder

Embryology

The urinary bladder develops from cloaca when the urorectal septum divides it into anorectal pouch posteriorly & urogenital sinus anteriorly. The urogenital sinus carrying with it the two ureters is the future bladder.

Anatomy

The urinary bladder is the reservoir of urine. It is a muscular hollow organ whose normal capacity is about 400ml in adult.

Surfaces of the urinary bladder.

The bladder has the following surfaces:

1-The superior surface (vault).

-It is covered with peritoneum.

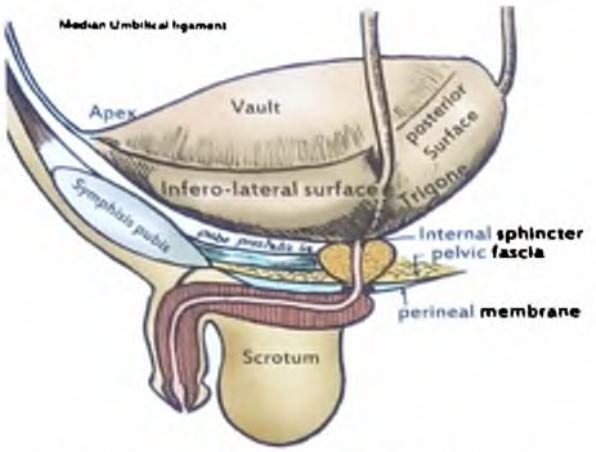
2-The two inferio lateral surfaces.

They rest on the pelvic floor.

3- Posterior surface.

-It is narrow surface above the base.

- In males, it is covered with peritoneum and related to loop of intestine in the rectovesical pouch, but in females, it is related to the ant. wall of the vagina.



Anatomy of the urinary bladder

4- Base: A triangular surface receives the ureter at its base and gives the urethera.

-Its outer aspect in males is related to seminal vesicles & termination of the vas deferens. Its inner aspect is called the trigone of the urinary bladder, which has a smooth stretched mucosa between the two ureteric orifices & internal urethral meatus.

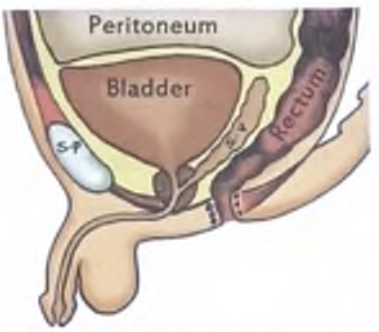
-The ureters pierces the base of the bladder and pass within its wall as the intramural parts of the ureter till they open in the lateral angles of the trigone as the two ureteric orifices .

Ligaments of the bladder:

1- Median umbilical ligaments:

The obliterated urachus joins the apex of the bladder to the umbilicus.

2- Two lateral umbilical ligaments. They are the obliterated distal part of the superior vesical artery (the two obliterated umbilical arteries).



Relations of urinary bladder

3- *The two true lateral ligaments*, they connect the urinary bladder to the lateral pelvic wall.

4- *The two true posterior ligaments*. They surround the veins emerging from the vesical venous plexus to the internal iliac vein.

Blood supply:

Arterial supply: from:

-*The superior vesical arteries* from the proximal patent part of the umbilical artery.

-*The inferior vesical artery* (only in males from internal iliac artery. In females, branches from the vaginal and uterine arteries supply the bladder).

Venous drainage:

It drains in vesical venous plexus on the infero-lateral wall that drains through the veins of the posterior ligament into the internal iliac veins.

Nerve supply:

1- Sympathetic supply.

From $T_{11, 12}$ & L_1, L_2. They are inhibitory fibres to the wall and stimulatory to the sphincter.

2- Parasympathetic supply:

From S_2, S_3 & S_4. They are stimulatory fibres to the wall and inhibitory to the sphincter.

Lymphatic supply:

Lymphatic drainage is mainly to the external iliac LNs.Some lymphatics (from the base) drain into the internal iliac LNs. From both groups, lymphatics reach the common iliac LNs.

Applied anatomy

1-The intramural part of the ureter acts as a valve that prevent the reflux of urine into the ureter because it is compressed by the bladder contraction.

2- When the bladder is distended with urine, it elevates the peritoneum from the lower part of the anterior abdominal wall so; part of its anterior surface is related directly to the anterior abdominal wall. This fact is very beneficial during percutaneous suprapubic cystostomy.

3- The urinary bladder is identified during operation by:

1) Midline cystic organ in the pelvis.

2) Not covered with peritoneum except its vault and the posterior surface in males.

3) It is surrounded with a plexus of vein.

4) It distends when saline is injected into the urethral catheter.

I- Congenital anomalies

1- Ectopia vesica (exstrophy).
2- Congenital anomalies of the urachus.
3- Marion's disease.
4- Congenital diverticulum.
5- Septate, bipartite & lobulated urinary bladder.

1- Ectopia vesica.

Aetiology:

Failure of the urogenital sinus to be supported by mesoderm anteriorly so, it ruptures in utero.

Pathology:

Types: There are two types:

I-Complete type: There are:

1) Absent anterior abdominal wall below the umbilicus.
2) Absent anterior wall of the bladder.
3) The post. wall of the bladder is pushed anteriorly by the intra-abdominal pressure which bulges through the defect with obvious two ureteric orifices dribbling urine continuously.
4) Wide separation of the pubic bones "they are connected with a long strong ligament".
5) The urethral sphincter is splitted & the rectal sphincter is usually lax.
6) The penis is small with complete epispadius. In females, the clitoris is cleft and the labia minora are separated anteriorly.
7) Bilateral undescended testis with bifid scrotum.
8) Umbilical and may be inguinal hernia.

II-Incomplete type :

The pubic bone is good united and the external genitalia are mostly normal.

Complications:

1- Recurrent ascending pyelonephritis "the commonest cause of death below 30 years".
2- Excoriation of the skin.
3- Phosphatic encrustation on the bladder mucosa.
4- Metaplasia and neoplasia of the bladder mucosa.

Incidence:

It is more common in males (♂:♀ =4:1)

Clinical picture:

-The condition is evident at birth.
-Other congenital anomalies may be present.

Investigations:

To diagnose other congenital anomalies e.g. plain X ray pelvis.

Treatment:

I-Cases diagnosed early: Closure of the bladder and pelvis is easiest in the 1st 48 hours.

-Correction of epispadius is achieved at the age of 2-3 years.

-Correction of incontinence is delayed for somewhile, until the bladder capacity increased.

II- Neglected cases or cases present with complications:

Urinary diversion e.g. ureterocolic implantation with cystectomy may be the only line of ttt.

2-Congenital anomalies of the urachus

A- Congenital urachal diverticulum:

-It is due to persistent patent proximal part of the urachus.

-It is attached at the apex of the bladder & predisposes to adenocarcinoma of the bladder.

-Treatment is surgical excision.

B- Patent urachus (cong.urinary fistula):

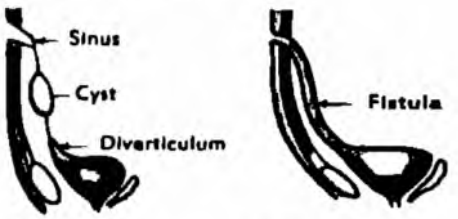
-Due to persistent & patent whole urachus.

-It causes congenital urinary fistula if associated with bladder outflow obstruction e.g. BNO.

-Treatment: excision of the fistula and treatment of the obstructive element.

C- Urachal sinus:

- Persistent patient distal part of the urachus.
- It causes mucous discharge per umbilicus.
- *Treatment*: excision.



Congenital anomalies of the urachus

D- Urachal cyst.

- Persistent patent middle part of the urachus.
- It presents as an intra-abdominal cystic swelling in the midline **below** the umbilicus moving from side to side not up & down.
- Treatment: excision.

3- Marion's disease

- It is congenital contracture of bladder neck.
- It may be due to congenital hypertrophy of internal urethral sphincter.
- It is treated with transurethral resection, which may lead to incontinence in females or retrograde ejaculation in males.

II- Diverticula of the urinary bladder

Aetiology:

I- Congenital.

1- Paraureteric diverticulum: due to rudimentary ureteric bud.

2- Urachal diverticulum: at the apex of the bladder (see above).

II- Acquired diverticulum.

1- Pulsion diverticulum: secondary to bladder neck obstruction & increase in the intraluminal pressure leading to trabeculation of the muscle coat so, the mucosa will bulge in-between. *The commonest site is Paraureteric site.*

2- Traction diverticulum: It is due to:

1- Traction by nearby fibrosis.

2- Traction by peritoneum in sliding groin hernias.

3-False diverticulum:

It occurs when pelvic abscess ruptures into the bladder and mucous membrane creeps to line its cavity forming a false diverticulum.

Pathology:

Pulsion diverticula are devoid of the muscle coat, so they are liable to stasis and complications.

Complications:

1- Stasis →recurrent cystitis. 2- Stone formation.

3- Paraureteric diverticulum may compress on the lower ureter →hydroureter, & hydronephrosis.

4- Malignant changes: in congenital diverticulum (adenocarcinoma) and in pulsion diverticulum. Carcinoma has rapid spread due to the thin wall.

Clinical picture:

I- Asymptomatic cases: Most cases of congenital diverticulum are symptomless.

II- Symptomatic cases:

A- History of the cause in acquired type e.g. SEP, BNO.

B- Double micturition: The patient feels a second desire for micturition after evacuation of the bladder especially when he stands up that is because the diverticula evacuate their content of infected urine on trigone after relaxation of the bladder initiating intense desire for micturition, which may be turbid *(pathognomonic).*

III- Picture of complications: cystitis, hydronephrosis.

IV- PR examination: - It may reveal the cause e.g. SEP.

- Bimanual examination rarely very large diverticulum can be felt.

Investigations:

1- Urine analysis: for pus cells & crystals.

2- Plain X ray: It may show stone in the diverticulum "stone in high lateral position".

3- IVU: It shows the diverticulum (lateral view should be performed).

4- Cystoscopy: It may show the mouth of the diverticulum as a black spot.

Treatment:

I- Asymptotic cases: No treatment.

II- Symptomatic cases:

A- Multiple small diverticulae: If the cause is corrected, They usually disappear spontaneously

B-Large diverticulum with narrow neck needs excision with correction of the cause. It can be excised through intraperitoneal or extraperitoneal approach. The latter may be extravesical or transvesical.

Treatment of problematic cases

Big diverticulum with senile enlargement of the prostate. If we removed the prostate first, secondary infection & haemorrhage may occur from the bed of the prostate and removal of the diverticulum later will be more difficult and if we removed the diverticulum first, the prostate will represent distal obstruction with possibility of urinary fistula. Therefore, the best is to operate on both at the same setting after endoscopic preparation of the diverticulum.

III- Trauma

Rupture bladder

Aetiology:

I- Precipitating trauma:

1- Closed trauma:

a) Blow or kicks in the suprapubic region especially when the bladder is distended e.g. Saturday night injury when a drunken person has a blow in the abdomen with the bladder full causing intraperitoneal rupture of the bladder.

b) Fracture pelvis: the sharp ends of bones are driven inward perforating the bladder → extraperitoneal rupture of the bladder.

2- Open trauma: stab, gunshots & instrumentations.

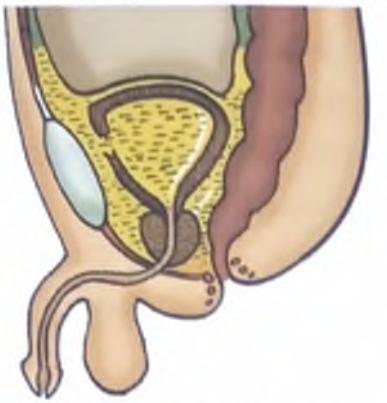
3- Spontaneous rupture: diseased urinary bladder may rupture on slight trauma.

II- Predisposing factors:

1- Distended urinary bladder.

2- Diseased urinary bladder weakened by ulcers or infection.

Pathology:



Extraperitoneal rupture

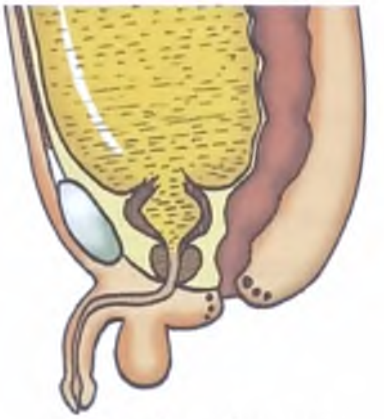
I- Extra peritoneal rupture (80%):

- Site: commonly in the anterior wall.
- It usually occurs due to fractured pelvis.
- Extravasation occurs in the perivesical & periprostatic space. If neglected, urine ascends up in the abdominal wall between the peritoneum & fascia transversalis.

II- Intra peritoneal rupture (20 %):

-Site: the dome of the bladder.

- It usually occurs due to blunt abdominal trauma.
- Extravasation occurs in the peritoneal cavity.



Intraperitoneal rupture

Complications:

1- Shock, & haemorrhage.

2 - Infection: pelvic cellulitis in the extraperitoneal type & peritonitis in the intraperitoneal type.

3- Associated injuries e.g. rupture urethera & other viscera.

Clinical picture:

I- The extraperitoneal type:

1- History of trauma: usually severe e.g. run over accident.

2- Shock : usually marked.

3- Pain & tenderness: suprapubic pain & tenderness, which remain localized for a long time then, spreads up the abdominal wall.

4- Desire of micturition: intense desire for micturition but the patient cannot void urine.

5- PR exam.: may feel the extravasated urine as a soft swelling around the prostate & bladder .

6- Catheterization: It usually yields small amount of urine and few drops of blood.

II- The intraperitoneal type:

1- History of trauma. e.g. abdominal trauma .

2- Shock is mild unless there are other injuries.

3- Pain & tenderness: suprapubic pain & tenderness, which soon becomes generalized in the abdomen (peritonitis).

4- Desire for micturition: No desire for micturition.

5- PR examination: may feel the extravasated urine as fullness in the recto vesical pouch.

6- Catheterization: It yields few drops of urine & blood.

Investigations:

1- Plain X ray: It may show fracture pelvis.

2- Abdominal U/S: It diagnoses rupture by failure of the bladder to distend with saline injected through a catheter and detects extravasation.

3- IVU: It shows **leakage** of dye from the bladder giving *flame shaped area* in extraperitoneal rupture **or** ***sun burst appearance*** **in** intraperitoneal rupture

Treatment:

I- First aid:

- Treatment of **shock i.e. bl. transfusion, IV fluid** ...etc.
- Introduce **a Foley's catheter.**

II- Definitive ttt:

A- Extraperitoneal rupture:

1- **Small tear & minimal extravasation: fix Foley's catheter in the** bladder plus broad-spectrum antibiotics, is usually all what should be done.

2- **Large tear with marked extravasation:**

- **Suprapupubic midline incision, exploration of the bladder** extraperitoneally, evacuation of extravasated urine, repair of the tear, fixation of a suprapubic cystostomy tube and drain the retropubic space (cave of retzius).
- **Management of a possible fracture pelvis as usual.**

B- Intraperitoneal rupture:

- Suprapupubic **midline incision, evacuation of the extravasated urine, sealing** the tear in the urinary bladder, closure of the peritoneum and explore of the bladder extraperitoneally .This is followed by fixation of a suprapubic cystostomy tube then drainage of retropubic space.

-Management of a possible other organ injury.

IV- Inflammation

Bilharziasis of the urinary bladder

Aetiology:

Causative organisms: Schistosoma haematobium.

Life cycle (see before).

Pathology:

The pathology occurs due to deposition of the ova in all coats of the bladder.

Site: The ova are deposited in all coats maximally in the submucosa especially in the trigone.

Mac:

The reaction of the bladder wall to ovideposition may be:

A- Hypertrophic.

1- Bilharzial tubercle: submucous nodule. It appears grayish white with pink halo around it.

2- Bilharzial granuloma: aggregated tubercles over which the mucosa may ulcerate.

3- Bilharzial polyps: It is bilharzial granuloma pushing the overlying mucosa in front of it.

B- Atrophic lesions:

1- Sandy patches are calcified ova that look under atrophied mucosa like sand under water.

2- Bilharzial ulcers: they are due to passage of ova (abrasion), ischaemia & sloughing of the mucosa by underlying fibrosis or due to falling of polyps.

3- Fibrous contractions, which may involve:

a- The prostate and internal meatus leading to bladder neck obstruction.

b- The whole bladder leading to contracted bladder.

C- Bilharzial carcinoma (see later).

MIC:

1- Submucosa. It shows bilharzial granulation tissues.

2- Mucosa: it shows epithelial hyperplasia that may lead to

a) Brunn nests: the hyperplastic epithelium buries itself under the surface to form a nest that will separate from the surface.

b) Cystitis cystica, when central necrosis and lysis occurs in Brunn's nests.

c) Cystitis glandularis: It develops when the transitional epithelium of the cystitis cystica undergoes metaplasia into columnar epithelium.

d) Squamous metaplasia may develop.

Complications:

I- General:

1- Anaemia. 2- Weight loss. 3-General weakness.

II- Local:

1- Recurrent infection & haematuria. 2- Bladder neck obstruction.

3- Stricture ureters. 4- Contracted bladder.

5- Hydroureter & hydronphrosis. 6- Stone formation.

7- Renal failure. 8- Carcinoma of the bladder.

Incidence:

It is more common in young adult male farmers.

Clinical picture:

I- History of contact with infected water.

II- Symptoms:

1- Terminal haematuria: It is an early symptom. It occurs as passage of ova is enhanced by the muscular contraction of the bladder at the end of micturition.

2- Pain: suprapubic pain especially during micturition.

3- Symptoms of complications.

III- PR exam: may reveal rectal polyp of intestinal bilharziasis.

Investigations:

See bilharzial stricture of the ureter.

Treatment:

I- Medical treatment: for all cases.

- Antibilharzial treatment.

II- Instrumental management: It is indicated in the following:

1- B. polyps: Cystoscopic fulgration.

2- Resistant B ulcer: Cystoscopic diathermy coagulation.

3- Stone: litholapaxy and stone extraction.

III- Surgical: Indications.

1- Resistant ulcer or polyps. suprapubic incision → excision.

2- BNO: wedge resection or Bonnin's operation.

3- Contracted bladder: ileocystoplasty.

Interstitial cystitis.

(Hunner's ulcer, elusive ulcer)

- Infection of the paravesical tissue (paracystitis) with secondary fibrosis & ulceration of the bladder.
- It is much more common in middle-aged females.
- The patient complains of diurnal & nocturnal frequency with suprapubic pain that isrelieved by micturition.
- Cystoscopy shows the characteristic linear or star shaped ulcer that appears on distension
- Overdistension of the bladder under anaesthesia for 2 hours is tried if fails partial cystectomy with ileocystoplasty may be needed.

Bladder neck obstruction

Definition:

Narrowing of the urinary bladder outlet. This term is applied to those patients who suffer from symptoms of senile enlargement of prostate without palpable prostate"Prostatism sans prostate".

Aetiology:

I- Congenital hypertrophy of the internal urinary sphincter (Marion's disease).

II-Acquired fibrosis of the bladder neck due to:

1-Bilharziasis: the commonest cause of BNO in Egypt

2-Chronic prostatitis in men and urethrotrigonitis in women.

Pathology:

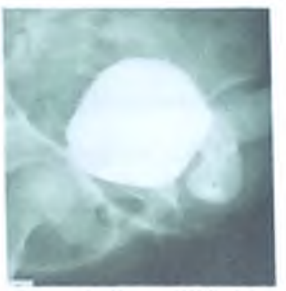
There is thickening and narrowing of the bladder neck causing obstruction to the urine outflow.

Complications:

General complications: Hernia, piles, & rectal prolapse due to excessive straining.

Local complications:

1-Trabeculation & diverticulae.
2-Recurrent cystitis.
3-Stone formation.
4-Bilateral hydroureter & hydronephrosis.
5-Recurrent pyelonephritis & stones.
6-Renal failure.



Funnel shaped bladder

Clinical picture:

The symptoms are similar to those of SEP but:

1-The duration is longer.
2-The patient is often younger.
3-Haematuria is less frequent.
4-Straining helps the stream of urine.

Investigations:

1-Urine analysis.
2-Kidney function tests.
3- IVP: It may show: *Funnel shaped bladder
*Function of both kidneys.
* diverticula, & hydroureter.

Treatment:

I-Instrumental:

-Dilatation only for early cases.

-Transurethral resection of thickened posterior lip of the internal meatus.

II-Surgical:

1-Transvesical resection of the posterior lip of the internal meatus.

2-Bonin's operation: V-Y sphincteroplasty.

Bonnin's V-Y sphincteroplasty

V- Bladder stones

Aetiology:

I- Source of stone:

1- Primary formed in the bladder (more common).

2- Migrating stone from kidney & ureter (less common).

II- Predisposing factors:

1- General factors. see before

2- Local factors:

a- Obstruction, BNO, & prostate.

b- Infection: cystitis & bilharziasis.

c- Urinary bladder diverticulum.

d- Foreign bodies, e.g. suture material.

Pathology:

Site: Free stones are usually resting over the base of the bladder.

Types: It may be oxalate, urate, phosphate or cystine stone. However, all types will acquire a rapidly increasing coat of phosphate especially in alkaline urine froming a big stone.

Complications:

1- Migration → retention of urine.

2- Infection: recurrent cystitis.

3- Obstruction: rarely, a big stone may obstruct ureteric orifices and causes hydronphosis.

4- Ulceration & haematuria.

5- Malignant change: in long standing stones.

Incidence:

It is more common in children & young adult males.

Clinical picture:

I- Asymptomatic case: some cases are asymptomatic discovered during routine investigations.

II- Symptomatic cases:

1- Frequency: diurnal more than nocturnal. This is because in standing position, the stones rest on the trigone irritating it while on supine position, it rests on the less sensitive posterior wall. When infection ensues, frequency becomes both diurnal & nocturnal (day & night) .

2- Pain: suprapubic & referred to the tip of the penis or perineum.

3- Difficulty if the stone obstruct the internal meatus

4- Strangury (bladder tenesmus): sense of incomplete evacuation of the bladder.

5- Features of complications e.g. haematuria, retention & pyuria.

III- PR exam. : -It may detect a predisposing factor e.g. SEP.

-A large stone can be rarely felt by bimanual examination.

Investigations:

1- Urine analysis: for RBCs, pus cells, B ova & crystals.

2- Kidney function tests.

3- Abdominal U/S: It is very helpful.

4- Plain X ray: almost all bladder stones are radio opaque due to the phosphate coat they appear in the centre of the pelvis but it may appears in abnormal high lateral position if the stone is:

1- In a diverticulum.

2- Over a tumour.

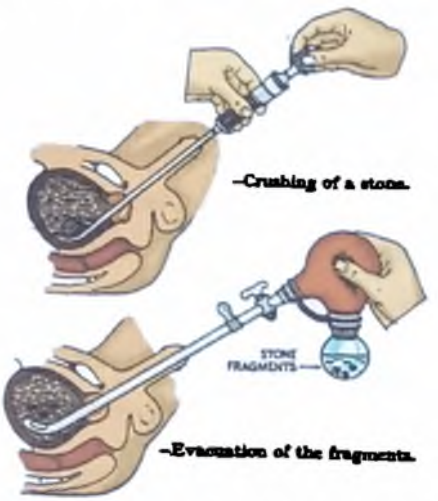
3- In the intramural part of the ureter.

4- Over suture material

5- IVU :(see before)

6- Cystoscopy:

- It detects the stone.
- It diagnoses a predisposing factor e.g. B cystitis.
- It detects associated lesions e.g. diverticulum.



Trans-uretheral litholapaxy

Treatment:

I- Conservative ttt:

It is rarely successful as most stones are large.

II- ESWL:

It may be used in bladder stone.

III- Instrumental ttt:

1- Crushing the stone (Trans-uretheral litholapaxy):

Crushing stones with a lithotrite then evacuation of small fragments with Bigelow's evacuator.

2- Electro hydraulic lithotripsy (EHL) or (USL): It is the treatment of choice in stones less than 3 cm. Stone fragments are removed with Bigelow's evacuator.

IV- Surgical ttt:

Indications: contraindications for instrumental.

1-distal obstruction: e.g. SEP or BNO.

2-Infection: urethritis, cystitis

3-Large stones (> 3 cm).

4- Multiple stones.

5- Too hard stones.

6- Too soft stone.

7- Stone formed on foreign bodies.

8- Stone in a diverticulum.

9- In children.

10- Failure of crushing

Procedure = Cystolithotomy

-Midline suprapubic incision and the bladder is reached extraperitoneally and opened. Stone is extracted & the bladder is closed with in two layers. The retropubic space is drained.

VI- Neoplasms

A- Benign neoplasm

1- (Villous papilloma)

Aetiology:

1- Primary tumours: Aniline dye workers.

2- Secondary tumour: surface implantation from renal pelvis or ureteric papilloma .

Pathology:

It is single or multiple sessile or pedunculated polyps with multiple frond like villi.

It is now considered as stage I grade I carcinoma.

Complications:

1- Haematuria: painless, periodic, & profuse panhaematuria.

2- Attacks of retention when papilloma arises near the internal meatus and arrest the stream by ball and valve mechanism.

Clinical picture:

Patients present with complications e.g. haematuria.

Investigations:

IVU: It shows small filling defect.

Cystoscopy: detects tumour & allows biopsy.

Treatment:

1- Endoscopic fulgration in a single papilloma.

2- Open (surgical fulgration): when papillomas are numerous and sessile.

3- Partial cystectomy: for enumerable papilloma crowded in a localized area and for big tumours.

B- Malignant tumours.

(Carcinoma of the bladder)

Classification:

It is the commonest urologic malignancy in Egypt.

In the past, we used to describe carcinoma of the bladder as bilharzial and non-bilharzial carcinoma, but now, the universal classification is according to the histological type as follow:

1- Transitional cell carcinoma 55 %.

2- Squamous cell carcinoma 40 %.

3- Adenocarcinoma 5%.

These figures are recorded by the registry of urology department at Cairo University hospital.

Aetiology:

I- Transitional cell carcinoma:

1- Aniline dye worker. 2- Phenacetin abuse. 3- Artificial sweetener.

4- Smoking. 5- Villous papilloma. 6- Cyclophosphamide.

II- Squamous cell carcinoma:

1) Bilharziasis. It is common in Egypt due to prevalence of bilharziasis.

Bilharziasis predisposes to malignancy of the bladder through:

1- Mechanical irritation theory: Repeated passage of B. ova causing irritation of the bladder wall leading to metaplasia and finally neoplasia. This theory is not accepted because:

a- The trigone is the commonest site of ovideposition, but, the rarest site for B. carcinoma.

b- Mechanical irritation by stone rarely induces carcinoma of the bladder.

c- Irritation of the colon by bilharzial ova rarely induces carcinoma.

2- Miracidial toxins theory: absorbed toxins from disintegrated ova are carcinogenic.

3- Tryptophan metabolism theory: Associated liver fibrosis and dysfunction leads to derangement of tryptophan metabolism with production of carcinogenic materials.

4- Alkaline sepsis theory (most accepted): Associated sepsis with production of ammonia leading to alkaline pH that induces phosphatic encrustation and squamous metaplasia .However, non-bilharzial alkaline cystitis is not precancerous.

2) Smoking. *3) Long standing cystitis.*

4) Long standing stones. *5) Ectopia vesicae.*

6) N.nitorso compounds contamination of vegetables & drinking water by nirates fertilizers leading to excretion of nitrates in urine, which are acted upon, by bacteria with liberation of N-nitroso compounds, which are carcinogenic.

III- Adeno carcinoma. It may develop on:

1-Urachal diverticulum 2-Cystitis glandularis 3-Mucous glands at the trigone.

Pathology:

Site.

1] TCC: The commonest site is the trigone 40 %.

2] SCC: The commonest site is the lateral and posterior wall. The trigone is rarely affected.

3] Adenocarcinoma: Commonly in the vault of the bladder at the site of the urachus.

Mac:

1- Transional cell carcinoma:

a) Papillary growth (90%). b) Raised plaque. c) Nodular. d) Ulcerative.

2- Squamous cell carcinoma:

a) Fungating nodular type: (85%). b) Ulcerative type 10%.

c) Diffuse infiltrating type 4%. d) Papillary type 1 %.

MIC:

1) Transitional cell carcinoma. 2) Squamous cell carcinoma. 3) Adenocarcinoma.

Spread:

1- Direct spread.

a- Intrinsic: infiltrating the wall of the bladder .

b- Extrinsic, to the surroundings e.g. rectum, vagina, uterus, prostate, pelvic floor & bones.

2- Lymphatic spread: To LNs on the wall of the bladder, then obturator, external iliac, internal iliac and common iliac LNS. Finally, spread occurs to paraaortic LNS.

NB. lymphatic spread is rare & late in bilharzial cases as fibrosis obstructs the lymphatics.

3- Blood spread is rare & late. mostly, to lungs & bone (vertebrae) , liver & brain .

Complications:

1- Haematuria.

2- Obstruction to: a) the ureteric orifice causing hydroureter, hydronephrosis & renal failure.

b) Internal meatus, causing retention of urine.

3- Infection: cystitis and ascending pyelonephritis.

4-Distant metastasis.

N.B. Uraemia due to obstruction & recurrent pyelonephritis is the leading cause of death.

Incidence:

Carcinoma of the bladder is the commonest urologic malignancy in Egypt due to prevalence of B.

1) TCC: It is common in old (> 60 y.) males (M: F = 3: 1). It is common in urban areas.

2) Squamous cell carcinoma: It is common in young adult (20-50 years) males (♂:♀= 4:1).

Clinical picture:

1- *History of Bilharziasis*, (In large number of cases).

2- *History of long standing cystitis*, which becomes more severe when carcinoma ensues.

3- *Haematuria:*

-It is more with TCC than SCC.

-It is painless & profuse in TCC ,but painful & less severe in SCC.

4- *Necroturia*: Passage of white pieces of necrotic tissue.

5- *Pyuria*: Passage of turbid urine with foul odor.

6- Pain: This may be:

a- Suprapubic pain referred to the tip of penis (cystitis pain).

b- Dysuria: due to encroachment on internal meatus.

c- Dull aching loin pain: due to encroachment on ureteric orifices.

d- Severe boring pain: due to infiltration of pelvic nerves.

7-Strangury: (bladder tenesmus): Sense of incomplete evacuation.

8- Frequency: (Nocturnal as well as diurnal) due to:

1) Irritation of the bladder wall by carcinoma.

2) Encroachment on the bladder capacity by the tumour.

3) Development of cystitis.

9- Swelling: which may be:

a) Suprapubic mass: Tumour mass may be felt by bimanual exam.

b) Loin mass: hydronephrotic kidney may be felt in the loin.

10- PR: Clinical staging (*Wallace classification*):

T_0 = No definite mass.

T_1 = Palpable mobile mass with no induration of wall around.

T_2 = Palpable mobile mass with induration of the bladder wall.

T_3 = Palpable mobile mass with extravesical spread.

T_4 = Fixed bladder mass.

Wallace classification

Investigations:

1- Urine analysis: for pus cells, RBCs, B. ova, necrotic tissue & cytology for malignant cells.

2- Kidney function tests.

3- Ultrasound. a) Abdominal U/S, can detects the tumour mass inside the bladder.

b) Transrectal U/S, is very beneficial to detect the degree of tumour invasion

4- Plain X ray: a) Calcified tumour.

b) Eaten up shadow of calcified bladder in Bilharzial carcinoma (***Makar's sign***).

c) Stone in high lateral position, (stone over the tumour).

5- IVU may show: - Irregular filling defect in the urinary bladder.

- Function of both kidneys.

- Hydroureter, & hydronephrosis.

6- Ascending cystography: when renal function is impaired.

7- CT, MRI & chest X ray: To assess operability.

8- Cystoscopy & biopsy: is mandatory.

Irregular filling defect

Differential diagnosis:

From other causes of haematuria.

From the ordinary cystitis with frequency, dysuria, pain, and even haematuria.

Treatment:

I- Operable case:

A- Transitional cell carcinoma.

1) Superficial TCC:

-Cystoscopic resection of the tumour with the underlying muscle. (Nd-YAG laser may be used) + intravesical chemotherapy or immunotherapy (BCG) to avoid recurrence .

-Regular follow up for 5 years to detect recurrence. If it occurs, radical cystectomy with urinary diversion is indicated.

2) Muscle invasive TCC: Total radical cystectomy + urinary diversion + radiotherapy. .Interstitial irradiation can be done using radioactive gold or external irradiation + chemotherapy. Combination chemotherapy includes MVAC (Methotrexate + Vinblastine + Adriamycin + cisplatinum) .

B- Squamous cell carcinoma: → Radical total cystectomy + urinary diversion.

NB. In radical total cystectomy, we resect:

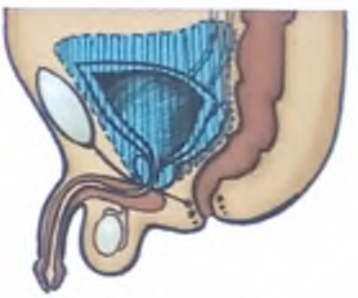
-The whole bladder & the overlying peritoneum.

-The lower 2 inches of ureters.

-The perivesical fat.

-The prostate, seminal vesicles & vasa deferentia in males or uterus, tubes & anterior vaginal wall in females.

-Internal, external & common iliac LNs as one mass.



Radical total cystectomy

NB. Pelvic exenteration (evisceration) = radical total cystectomy + excision of the rectum & perirectal tissue with terminal wet colostomy is done for tumours invading the rectum. It is rarely performed as it carries high morbidity & mortality.

Inoperable cases:

I-Resectable tumor:-

Palliative resection with urinary diversion.

II-Irresectable tumors:-

1-Urinary diversion + radiotherapy + chemotherapy

2-Messures to relieve pain.

a-intrathecal injection of morphia or better alcohol. Pain is relieved for few months.

b-Presacral neurectomy or lat. cordotomy to interrupt the spinothalamic tracts on both sides.

Urinary diversion

To divert the ureters and urine flow away from the bladder (diseased or resected).

Methods:

I-Incontinent diversions

A-Ureterocutaneous anastomosis (ureterostomy).

The ureters are mobilized and opened at the skin.

Advantages:

Easy rapid operation, suitable to unfit patients with bad renal function.

Disadvantages:

1-Soiling of the clothes, and excoriation of the skin.

2-Ascending infection.

Ureterostomy

B-Ileal or colonic conduit: implantation of the ureters into an isolated loop of ileum or colon that is closed from one side and anastomosed to skin at the other side.

Advantages:

1- No or minimal urine leak.

2- No or little chance of ascending infection.

Disadvantage:

1- Soiling is not completely avoided.

2- Hypercholaremic acidosis may rarely develop.

3- Time consuming operation.

Ileal conduit

II- Continent diversions:

A- Continent diversions using the anal sphincter:

1-Ureterocolic implantation: Ureterosigmoidostomy.

The ureters are implanted in the sigmoid colon, which will serve as a reservoir for both urine & stool.

Advantages:

a- The patient is continent.

b- No excoriation & soiling.

Disadvantages:

a- Ascending infection.

b- Hypercholaremic acidosis due to excessive absorption of Cl^- from the colon. If the kidney function is good, it can re-excrete it again.

c- Stenosis of the stoma.

Ureterocolic implantation

Rectosigmoid bladder

d- Cancer colon in 30% after 10 years.

This operation is limited to fit patient with good renal function.

2- Rectosigmoid or rectal bladder:

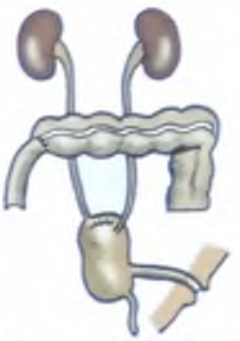
Terminal left iliac colostomy is done and the upper end of the rectum is closed, then the two ureters are implanted into the rectum, which will serve as urine reservoir. This is because colostomy is better and easier to be cared than ureterostomy.

Cock's pouch

B- Continent diversions with abdominal stomas

1- *Cock's pouch.* an isolated loop of ileum is intussuscepted into itself in order to design a valve, then one end is closed. The other end is anastomosed to the skin and the two ureters are implanted into it. This pouch evacuates only when the patient admits a catheter into it (clean intermittent self catheterization CISC).

2- *Ileocaecal bladder*. The caecum & terminal ileum are isolated. The caecum is closed and into it, the two ureters are implanted. The terminal ileum is opened to the skin. The ileocaecal valve opens only when the patient introduces a catheter (CISC).

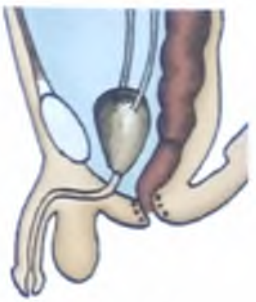


Ileocaecal bladder

C- Continent diversions using uretheral sphincter

=Orthotopic bladder

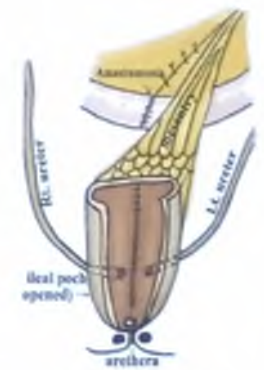
1- *Recto vesico urethroplasty*: The upper part of the rectum is isolated. Its upper end is closed, the two ureter are implanted into it and its lower end is anastomosed to the urethra or the bladder neck. The continuity of the gut is restored by anastomosing the colon to the lower rectum.



Recto vesico-urethroplasty

2- *Ileal or colonic orthotopic bladder :*

A suitable loop of ileum or colon is isolated and fashioned to form a pouch which is then anastomsed to the urethra to substite the resected bladder



Ileal orthotopic bladder

Prognosis:

The 5 years cure rate for operable cases are about 32 % after radical cystectomy.

The urethra

Anatomy

Male urethra:

The male urethra consists of:

1-**prostatic urethra**: This part runs inside the prostate.

2-**Membraneous urethra**: This part runs in the deep perineal pouch.

3-**Penile urethra**: This part runs in the corpus spongiosum and opens in the tip of penis.

-The prostatic and membranous urethra constitutes the intrapelvic urethra, and the penile urethra represents the extrapelvic urethra.

-The urethra has two fossa:

1-Intrabulbar fossa in the bulb of the penis.

2-fossa navicularis: inside the glans penis.

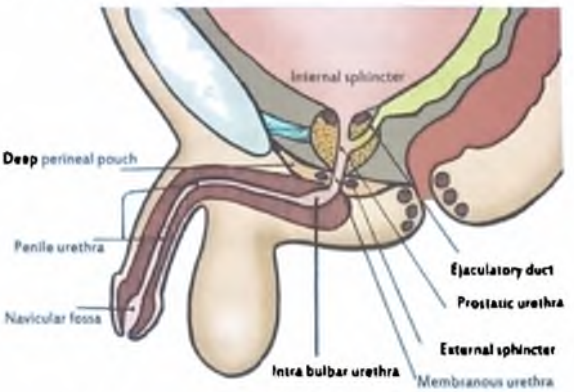
They are common sites of arrest of stones

The epithelial lining:

1-Prostatic urethra lined by transitional epith.

2-Membranous urethra lined by pseudo-stratified columnar epith.

3-The penile urethra is lined by stratified columnar epith that changes into stratified squamous epith (skin) near its end .



Anatomy of the male urethra

*The widest part is the prostatic urethra.

*The membranous urethra is the weakest part and together with the external meatus are the narrowest segments in the urethra.

I- Congenital anomalies

1-Phimosis: congenitally long & narrow prepuce (cannot be retracted over the glans) with a very narrow opening. Retention of smegma may occur leading to inflammation of the glans (balanitis).

2-Paraphimosis: the prepuce is retracted and tightly compresses on the veins & lymphatics of the distal penis. It may end in gangrene of the glans if not treated.

3-Meatal stenosis (pinhole meatus):

-It causes backpressure on the bladder.

-It is treated with meatotomy & dilatation.

4-Congenital urethral diverticulum.

5-Hypospadias

Definition:

A common congenital anomaly in which, the urethra opens on the ventral aspect of the penis.

Aetiology:

Glanular hypospadias is due to failure of canalization of the glans. The other types are due to failure of fusion of the two urethral folds so, the urethral groove remains opened to the ventrum of the penis.

Pathology:

Types:

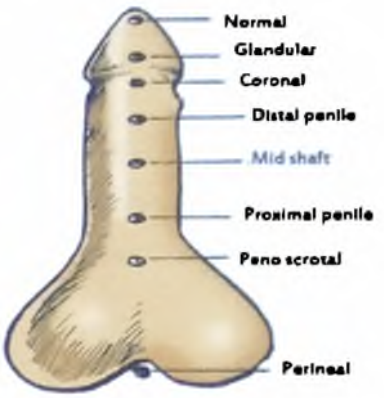
1-*Glanular*: the ectopic opening at the ventrum of the glans.

2- *Coronal*: the ectopic meatus opens at the coronal sulcus.

3-*Penile*: the ectopic meatus opens at the ventral aspect of the shaft of the penis.

4- *Penoscrotal*: the ectopic meatus opens at the junction of the penis with the scrotum.

5- *Perineal*: the ectopic meatus opens between the two halves of a bifid scrotum into perineum.



Types of hypospadias

-Pathological changes:

- The ectopic meatus lies on the ventral aspect of the penis and is usually stenosed.
- The corpus spongiosum distal to the ectopic meatus is fibrosed and forms a fibrous band that is called chordee, which causes ventral bowing of the penis.

-The prepuce is not complete on the ventral aspect like a hood over the glans (Hooded prepuce)

Complications:

1-Ventral curving of the penis, which causes difficulty in micturition and impotence.

2-Sterility: failure of proper insemination in the posterior fornix of the vagina. In addition, ventral curvature of the penis interferes with proper erection & intromission.

3-Psychological problems.

Treatment:

1-Glandular hypospadias.

- Corrected by Meatal Advancement and GlansPlasty Incorporated (MAGPI)

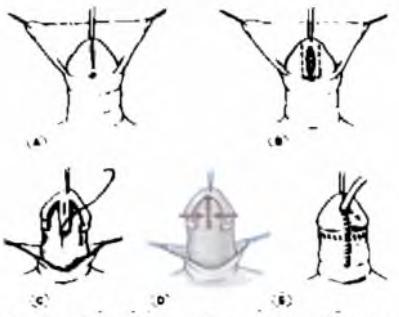
2-Other types:

After 6 M age according to penile size. Better before the child is oriented with genetalia.

- Distal hypospadias : - one stage with correction of chordee

-Tubularised Incised urethroPlasty (TIP) of Snodgras .Two lateral and one-midline incisions of urethral plate and a neourethra is fashioned around catheter.

- Proximal hypospadias: -two stages
  - The first for correction of chordee.
  - The second stage with preputial flaps (Duckett) or grafts to form neourethra.



Tubularised Incsed urethroPlasty

7-Epispadias

-It is due to abnormal opening of urethera on the dorsum of the penis, which appears spilt. Commonly associated bladder exstrophy with dorsal curvature of the penis.

-It is treated with plastic reconstruction.

II- Trauma

Rupture Urethra

Aetiology:

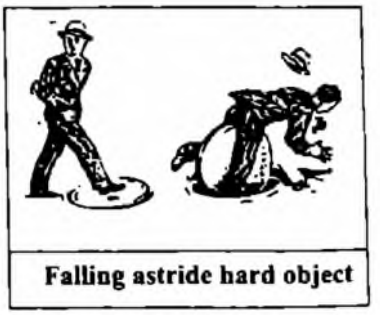
I-Direct trauma:

1-Direct trauma to the perineum e.g. Kick or falling astride hard object. This will crush the bulbous urethra against the pubis.

2-Instrumental injury, usually injure the posterior urethra.

II- Indirect trauma:

Major trauma fractures the pelvic rami, that in turn may tear the intrapelvic urethra.



Falling astride hard object

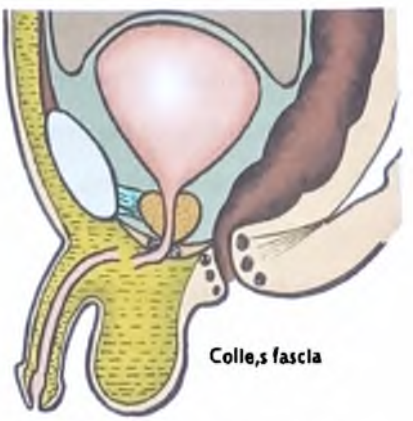
Pathology:

Types:

1-Extrapelvic rupture:

-The injury involves penile urethra at the bulbous part.

-If extravasation occurs, it will be in the superficial pouch, and then extends to the scrotum & penis and if neglected, it may extend to the anterior abdominal wall behind Scarpa's fascia. It does not descend to the thigh due to fusion between the superficial fascia and deep fascia of the thigh along a transverse line, which extends to the level of saphenous opening.

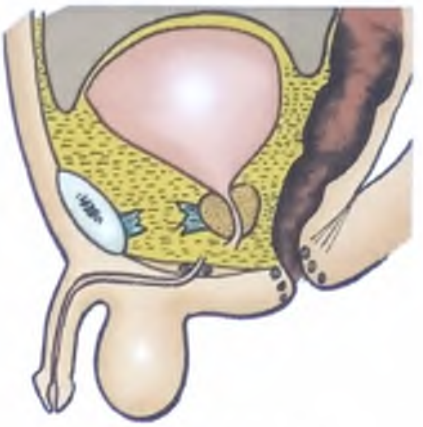


Extrapelvic rupture

2-Intrapelvic rupture:

-The injury usually involves the junction of the prostatic and membranous urethra.

-If extravasation occurs, it will be in the periprostatic & perivesical space, and if neglected, it extends to the abdominal wall between the fascia transversalis & peritoneum (deep).



Intrapelvic rupture

Extent:

1-Mucosal tear.

2-Incomplete tear.

3-Complete tear.

4-Complete tear with torn puboprostatic ligament and the prostate with the neck of the bladder are displayed away with no fixation (floating prostate) .A gap is present between the two ends of the torn urethera. It occurs only in the intrapelvic rupture.

Complications:

General:

1-Shock, which is more, marked in the intrapelvic urethra.

2-Associated injury of other organs e.g. extraperitoneal bladder rupture.

Local:

1-Infection.

2-Stricture urethra.

3-Incontinence of urine: if the urethral sphincters are injured in intrapelvic type.

Clinical picture:

1-Extrapelvic rupture is characterized by:

1-History of trauma to the perineum.

2-The shock is absent or minimal.

3-Perineal haematoma & pain
4-Bleeding per urethra
5-Retention of urine
} diagnostic triad

*Due to spasm of the sphincter.

*The bladder is felt distended with tense desire to micturition.

*It lasts few hours before extravasation takes place.

6-Extravsation of urine:

After few hours, swelling appears in the perineum, scrotum, penis, and finally in the anterior abdominal wall if neglected.

7-PR examination reveals no abnormality & the prostate is in its place.

II-Intrapelvic rupture:

1-History of major trauma (e.g. run-over accident).

2-Shock is usually marked.

3-Fracture pelvis may be evident with pain.

4-Few drops of blood may come from the external meatus after sometime.

5-Retention of urine. It lasts for a long period.

6-Extravasation of urine.

When it occurs, a suprapubic swelling appears and a cystic swelling is felt by PR exam.

7-PR examination: it reveals:

*The extravasated urine in the perivesical space.

*Non palpable or upward displaced prostate (floating prostate).

Investigations:

1-Plain X ray: to detect fracture pelvis.

2-Urethrography: detects the site of rupture and delineates the extravasation.

Treatment:

I-First Aid:

-Correction of shock if present.

-Instruct the patient not to try to void urine.

-Catheterization is contraindicated, as it may compound the damage and introduce infection.

There are two schools:

I-School of immediate repair.

Catheterization is tried in the operating theatre, under complete aseptic conditions. with all circumstances prepared for operation.

1-If the catheter succeeded to enter the bladder, this denotes partial tear in the urethra. The catheter is then fixed for 2 weeks together with full dose of broad spectrum antibiotics.

2-If the catheter fails to pass, this means arrest at the site of tear and accordingly:

A-Extrapelvic tear: →suture anastomosis is done.

- Open at the tip of the catheter, suture the torn ends then advance the catheter to the bladder and fix it for 3 weeks.

B-Intrapelvic rupture → sutureles anastomosis is done:

- Suprapubic midline incision is performed and the bladder is opened. A urethral dilator is advanced through the internal meatus. Another dilator is advanced through the external meatus, and both are moved until a click is elicited when the two dilators touch each other. The dilator in the urethra is advanced to the bladder & ordinary catheter is fixed to it

-The dilator to pulled out to bring the catheter out of meatus to which a suitable Foley's catheter tip is fixed. Again, the ordinary catheter is pulled from the bladder to advance Foley's catheter. The balloon is inflated and Foley's catheter is fixed for about 3 weeks.

-This method is called *Railway Method*. The procedure ends by suturing the torn puboprostatic ligament and suprapubic cystostomy.

-Fixation of the possible pelvic fracture by extenal fixator.

-Ascending cryptogram is performed to ensure integrity of the bladder.

-After 3 weeks remove the catheter & perform retrograde urethrography to detect stenosis. - If there is stenosis, repeated dilatation or visual internal urethrotomy VIU (urethroscopic division of fibrous tissues) for short strictures Or open urethroplasty for long ones.

II –<u>School of delayed repair.</u>

-No attempt is done for early cathertization, but only, a suprapubic cystostomy is done with:

*Incision drainage of perineal haematoma in extrapelvic rupture.

*Fixation of pelvic fractures in intrapelviclesions together with large dose of antibiotics.

-Urethral repair is delayed to a later date where urethroplasty is performed.

-The incidence of complications such as infection, incontinence, impotence and stricture are much less often and less severe than if immediate repair is carried out.

The Prostate

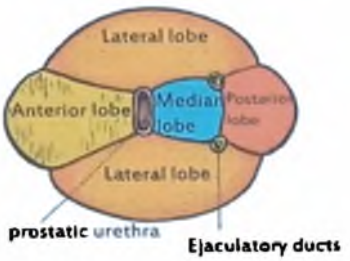
Anatomy of the prostate

- It is a small cone shaped gland that lies at the base of the bladder, resting over the pelvic floor.
- It has upper, anterior, posterior & two infero-lateral surfaces and an apex, which is directed downward.
- It has two fibrous capsules:
  * The true capsule due to peripheral condensation of its fibrous tissue stroma.
  * Prostatic sheath, a fibrous sheath surrounding the prostate derived from the pelvic fascia.

  Between the two capsules, lies the periprostatic venous plexus.
- The base (upper surface) is pierced by:
  1- The urethra that begins from the bladder neck, traverses the prostate, and comes out from the anterior surface just above its apex.
  2- The two ejaculatory ducts, which open into the prostatic urethra.

Anatomical lobes of the prostate

1- **The median lobe:** - It is the part of the prostate wedged between the urethra anteriorly and the two ejaculatory ducts posteriorly.
2- **The right & left lobe:** on both sides of the median lobe.
3- **The anterior lobe:** The part lying in front of the urethra and connects the two lateral lobes anteriorly.
4- **The posterior lobe:** The part behind the median lobe and connects the two lateral lobes posteriorly.



Cut section of the prostate

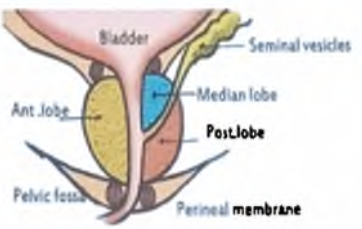
Recent classification of the prostate.

The prostate is divided into:

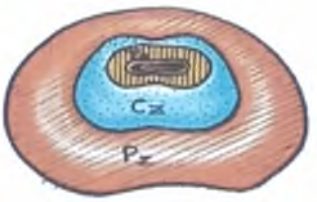
1- The peripheral zone (PZ):
- It lies on the periphery of the gland mainly posteriorly.
- It is the zone of carcinoma.

2- The central zone (CZ):
- It lies behind the urethra and in front of two ejaculatory ducts (corresponding to the anatomical median lobe).

3- The peri-urethral transitional zone (TZ):
- It surrounds the urethra.
- It gives rise to most of benign prostatic hyperplasia.



Anatomical lobes of the prostate



Zones of the prostate

Blood supply:

Arterial: from the inferior vesical, middle rectal & internal pudendal arteries.

Venous: into the peri prostatic venous plexus that drains into the internal iliac veins.

Nerve supply: From the pelvic plexus.

Lymphatic drainage. Lymphatics drain into the internal iliac LNS.

Physiology

- The growth of the prostate is under the control of testosterone, which is converted into the more active ingredient 5 dihydrotestosterone by the enzyme 5 alpha reductase enzyme in the prostate.
- The prostate specific antigen (PSA) is a glycoprotein present only in the prostate:
  * Its upper normal limit is about 4 nmol/ml.
  * Its serum level increases in benign prostatic hyperplasia & cancer prostrate.

Benign prostatic hyperplasia (BPH)

= Senile enlargement of the prostate (SEP).

Aetiology:

1-Hormonal imbalance: In old age, there is gradual but significant decrease in serum testosterone. However, the level of estrogenic steroids does not decrease equally. This imbalance causes enlargement of the prostate.

2-Neoplastic theory: It considers BPH as a benign tumour.

3- Inflammatory theory (not accepted):

It is based on appearance of chronic inflammatory cells e.g. lymphocytes in the stroma.

Pathology:

Site:

BPH usually arises from the periurethral transitional zone (TZ) and central zone (CZ). This causes enlargement of the anatomical lateral and median lobes. Rarely the anterior lobe but never in the posterior lobe (PZ), which is the lobe of carcinoma.

MAC

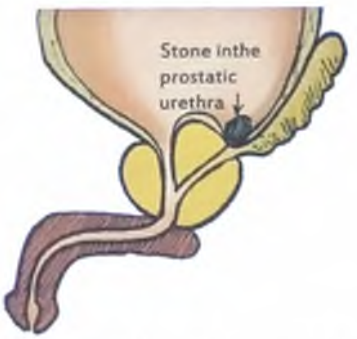
The prostate.

-There is symmetrical enlargement of the gland.

-The hyperplastic tissue (adenoma) enlarges and compresses the peripheral zone, so it has two capsules.

* False capsule from the compressed prostatic tissue.
* True capsule of the adenoma (the hyperplastic tissue)

The cut section is grayish in colour.



Median lobe enlargement

The urethra.

- It is elongated & narrowed.
- It may become tortuous with angulation.
- When the median lobe enlarges, it projects inside the bladder stretching the internal sphincter and everts the urethral mucosa into the bladder.

Lateral lobes enlargement

The urinary bladder.

- The obstructed outflow causes trabeculation of the wall with pulsion diverticula.
- The enlarged prostate projects into the bladder forming a post-prostatic pouch behind it in which residual urine stagnates.

MIC:

Changes are more or less similar to that occur in mammary dysplasia in the form of:

1- *Adenosis:* increased number of acini.

2- *Epitheliosis:* increased number of epithelial cells lining the acini forming multiple rows.

3- *Papillomatosis:* the hyperplastic epithelium projects in the lumen → papillary projections.

4- *Fibrosis:* there is hypertrophy of the fibromuscular stroma.

5-*Cyst formation*: Small cysts form due to hyaline degeneration of secretions & desquamated cells inside the acini forming what is called corpora amylacia.

6- *Round cell infiltration*: e.g. lymphocytic infiltration of the stroma.

There are 5 microscopic types of BPH according to the predominance of one or more element:

1- Adeno fibro myomatous.
2- Adeno fibromatous.
3- Adenomatous.
4- Fibromyomatous (FM).
5- Fibrous.
6- Muscular

Microscopic picture

Complications:

I- General.

1- Renal failure.

2- Complications of straining e.g. hernia, piles, & rectal prolapse.

II- Local.

1- Urethra:

a- Retention of urine: acute & chronic.

- Acute retention due to congestion & oedema at the bladder neck due to excess work, worry, weather (cold), water, wine & women.

-Chronic retention and may be retention with over flow.

b- Haematuria.

-More with median lobe enlargement.

-It may be mild from ruptured small veins but may be severe due to rupture of congested tortuous veins at the bladder neck (vesical piles) that occur due to compression of the prostatic venous plexus by enlarged prostate.

2- Bladder.

- Recurrent infection (cystitis) due to stasis.
- Stone formations due to both stasis and infection.

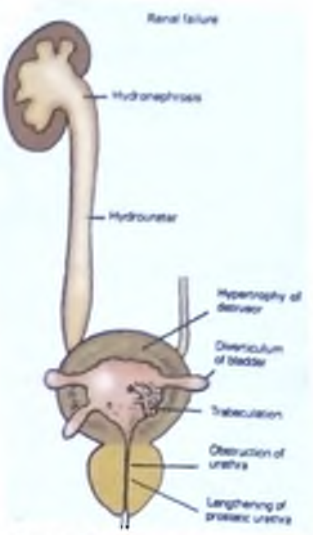
Stones usually rest in post-prostatic pouch.

-Pulsion diverticulum due to increased intraluminal pressure pushing the mucosa between the trabeculated musculosa.

3- Ureters & Kidneys:

-Hydroureter & hydronephrosis.

- Recurrent pyelonephritis.
- Stone formation.



Complications of SEP

Incidence:

- BPH affects 50% of males above 50 years. Never before the age 40 years.

Clinical picture:

Symptoms: The patient may have:

I- Silent prostatism or asymptomatic cases: (95%).

Most cases are asymptomatic and discovered during routine examination or investigations.

II- Uncomplicated prostatism:

1) Frequency: It is the earliest symptom. It is due to:

1- Eversion of the urethra into the bladder over the enlarged median lobe.

2- Stretching the internal sphincter, so, drops of urine may escape to the urethra initiating intense desire for micturition.

3- Congestion of the trigone and irritation by the residual urine.

4- Encroachment on the bladder capacity by huge prostate.

5- Polyuria due to renal impairment.

At first it is more nocturnal than diurnal. This is because of nocturnal pelvic congestion due to warmth, parasympathetic predominance and loss of muscular pump. Later, it becomes nocturnal & diurnal when cystitis and stones develop.

2) Difficulty in the acts: It means

1) Difficulty to start micturition.

The patient learns to relax and wait for few seconds to start the stream.

2) Difficulty to maintain the act, the stream is weak, interrupted and may be forked. If the patient strains, this may arrest the stream because straining increases the congestion & obstruction, and increases the kink of the urethra. In addition, in median lobe enlargement, the enlarged lobe acts as a ball & valve mechanism.

3) Difficulty to terminate the stream (post–voiding dribbling) due to weakness of the internal sphincter.

3) Sexual troubles.

In the early disease, there is increased libido. Later, there is impotence and decreased libido.

III- Complicated cases:

Many cases remain silent until present with complications e.g. haematuria, retention, infection or renal failure.

Signs:

General:

Signs of general complications e.g. renal failure & hernia

PR. examination ; the prostate is felt.

1) Symmetrically enlarged.
2) With Smooth surface.
3) Soft to firm in consistency.
4) Sulci are exaggerated.
5) Rectal mucosa slides over it.
6) A Sound in the urethra can be felt.

N.B. In isolated median lobe enlargement, it bulges in the bladder and is not felt on PR. exam.

Investigations:

I- Laboratory:

1- Urine analysis: for RBCs, pus cells & crystals.

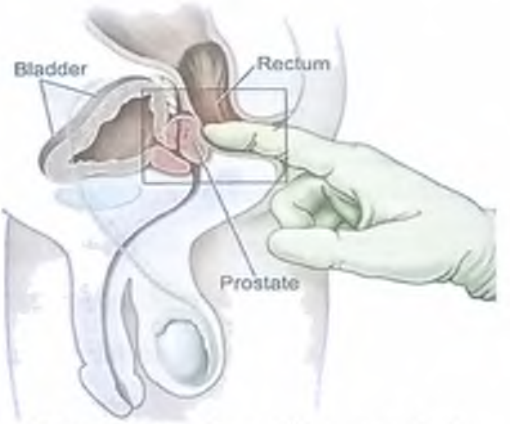
2- Kidney function tests:

II- Imaging studies:

1- Ultrasound (U/S):

1- Pelvi-abdominal U/S:

- shows the enlarged prostate.
- detects back-pressure on the kidneys.
- measures the residual urine.



PR examination of the prostate

2- Transrectal U/S (TRUS) is of great importance to differentiate BPH from prosthetic ca.

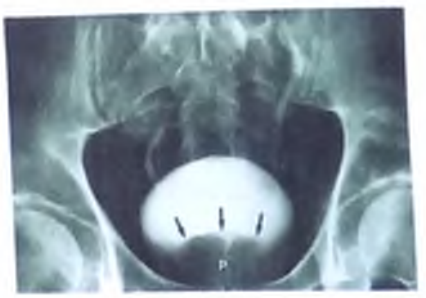
2- *Plain X-ray:* for associated stones.

3- *IVU:* It shows.

1) The function of both kidneys.

2) The enlarged prostate appears as :

- Elevation of the bladder base above the symphysis pubis.
- Smooth semicircular (median lobe enlargement) or butterfly (lateral lobes enlargement) filling defect in the base of the bladder.
- Residual urine in the post-voiding film.



Smooth filling defect at the bladder base

III- Uroflowmety: < 15 ml/sec means bladder outflow obst.

IV- Renal isotope scanning.

VI- Cystourethroscopy:

- It excludes other bladder pathology or complications.
- It excludes carcinoma of prostate.

VII- Preoperative investigations: to assess fitness for operation.

Treatment:

I- Conservative (Medical) treatment.

A- Moderation of life: avoid excess work, worry weather (cold), water, wine & women.

B- Drugs:

1- Uroselective alpha-blockers (alfazocin): They block alpha-adrenergic receptors in the urethra decreasing its tone.

2- 5 alpha reductase inhibitors (Proscar): It inhibits 5-alpha reductase enzyme and diminishes the size of prostate markedly .It improves most symptoms.

3- Decongestants: given as rectal suppositories to decrease congestion & oedema.

C- Control & treatment of any complications e.g. infection or retention.

D- Regular flow up: by uroflowmetry, TRUS, and urine analysis.

II- Surgical treatment:

Indications:

1- Prostatism disturbing the patient life (marked frequency & difficulty).

2- Residual urine more than 200 ml.

3- Complicated prostatism.

a- More than one attack of urine retention. b- Haematuria

c- Recurrent cystitis. d- Hydroureter, & hydronephrosis.

e- Other complications need surgery e.g. stones, & large diverticula.

Routes (Types) of prostatectomy (Prostatic adenectomy):

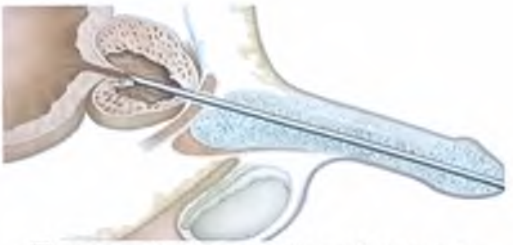
A- Transurethral (endoscopic) prostatectomy.

1- Transurethral resection of prostate (TURP:).

excellent method for selected cases :

* With enlargement < 80 gm.
* With median lobe enlargement.
* With fibrous prostate.

- The prostate is removed under vision with a resectoscope



Transurethral resection of prostate

2- Transurethral Microwave Thermotherapy (TUMT):

- Using a microwave-generating catheter, the microwaves travel only short distance and produce local heating of the tissues.
- It is a painless, rapid & easy procedure but the relief of symptoms is temporary.

3- Visual laser ablation of the prostate (VLAP):

Using laser energy to evaporate the obstructing adenoma tissue. It is bloodless procedure but the recurrence is commoner than TURP.

B- Transvesical:

Indications

1- When there is other bladder pathology needing surgery e.g. stone or diverticula.

2- In two stage prostatectomy.

Techniques.

1. *Fryer's technique:* blind enucleation of the prostate and bleeding is arrested using pack whose end is brought out through the wound and removed 48 hours later.
2. *Thompson Walker's technique:* blind enucleation then arrest haemorrhage by sutures under vision using special illuminated retractor.
3. *Harris technique (Trigonization),* after blind enucleation. The trigone is sutured to the urethra around a catheter using special boomerang (Harris) needle.
4. *Wilson Hey's technique (Trigonectomy),* after enucleation, the raised shelf of the trigone is excised to provide a better drainage of the prostatic bed into the bladder.

In all types, a suprapubic cystostomy tube is inserted and saline wash may be applied to prevent clotting of the blood.

C- Retropubic approach (Milin's prostatectomy):

The prostate is explored and removed through the retropubic space without opening the bladder.

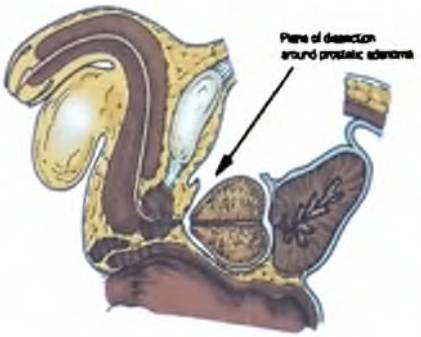
Advantages:

1- The bladder is not opened, no need for suprapubic cystostomy & rapid convalescence.

2- Excision & haemostasis are done under vision.

Contraindications:

1. Other bladder pathology needing surgery.
2. Obese persons with deep retropubic space.
3. Previous operations through the retropubic space.



Retropubic approach

D- Perineal approach: Young's prostatectomy.

It needs great experience in the region with high incidence of injury to the rectum, external urethral sphincter and nerves to the corpora cavernosa. In addition, urethral fistula is common so, it is rarely done nowadays.

Complications of Prostectomy.

I-Intra-operative complications:

1-Shock.

2-Haemorrhage (1ry).

3-Injury to bladder, external sphincter or adjacent structures.

4-Difficult enucleation of the prostate especially in fibrous type.

5-TUR syndrome due to excessive absorption of the irrigating fluid (glycine).

II-Postoperative complications:

1- Reactionary & 2ry haemorrhage.

2- Infection that may lead to epididymo-orchitis, therefore, some surgeons prefer to ligate the vas at the neck of the scrotum after enucleation of the prostate.

3- Incontinence (1%) due to injury of the external sphincter.

4- Obstruction (retention) due to:

-Fibrosis in the bladder neck.

-Missed lobule.

-Undiagnosed carcinoma of the prostate.

5-Impotence (5%) due to:

- Injury of the nerves to corpora cavernosa.

- Loss of the prostatic mechanical support of the base of the penis.
- Congested prostate during sexual excitation, compresses the periprostatic venous plexus into which the dorsal vein of penis drains and hence it helps erection.

6-Sterility (60%): due to retrograde ejaculation into the bladder (dry ejaculation) due to loss of internal sphincter mechanism.

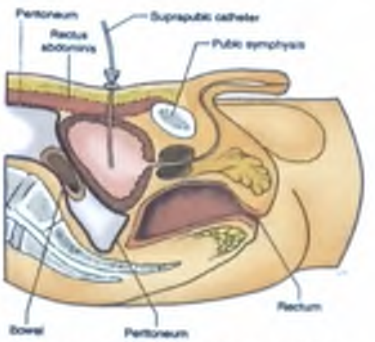
7- Ostitis pubis: superficial inflammation of the pubic bones that may lead to pain & limping. It resolves spontaneously.

8- General post-operative complications e.g. pulmonary complications.

III- Treatment of complications

e.g., retention, haematuria etc. see later.

N.B. Two-stage prostatectomy is resorted to, when urethral catheterization fails even with metallic catheter. A suprapubic cystostomy is done (first stage) and the patient is prepared for prostatectomy (2nd stage).



Percutaneous suprapubic cystostomy

Cancer Prostrate

Aetiology:

Prolonged androgenic stimulation:

Predisposing factors:

1-Family history of the disease. It is more in the first-degree relatives of cases.

2-Race: It is commoner among Africans & American and rare in the Asians.

3-Age: the incidence increases with age. It is very rare below 50 years.

4-Diet: fatty diet & red meat are incriminated while, carrots and cereals appear protective.

Pathology:

Site:

75% of tumours arise from the peripheral zone (posterior lobe).

MAC:

Hard non-capsulated irregular mass having grayish cut section with gritty sensation and areas of haemorrhage and necrosis.

MIC:

-Commonly *adenocarcinoma* with its different types.

-Columnar cell carcinoma forming malignant acini.

-Spheroidal cell carcinoma: Scirrhous, encephaloid & anaplastic carcinoma.

-Rarely, *transitional cell carcinoma* from the prostatic urethra.

Spread:

I- Direct spread.

Intrinsic: Early: upward towards the seminal vesicles and base of the bladder. In addition, infiltration of the lateral lobes is early.

Late: to the prostatic urethra.

Extrinsic: To the surrounding structures.

1- The rectum: it is late due to fascia of Denonvillier.

2- Ureter. 3- Sciatic nerve.

4- Iliac veins.

II- Lymphatic spread:

To the internal iliac LNs →common iliac LNs → para-aortic LNs →Thoracic duct → Virchow's LNs.

III- Blood spread:

Early & extensive to bone (90% in the pelvic bone, lumber vertebra & the head of the femur). It may be due to venous communication between the prostatic plexus and these bones.Bone metastasis are usually osteosclerotic. In addition, blood spread to the lungs is common.

Staging:

TNM staging

Tumour (T):

T_0 = No clinical abnormality.

T_1 = Palpable nodule in one lobe.

T_2 = Diffuse carcinoma.

T_3 = Extension to the seminal vesicles.

T_4 = Fixed tumour to the other tissues.

Nodes (N):

N_0 = No evidence of LN involvement.

N_1 = involvement of one regional LN.

N_2 = involvement of several regional LNs.

N_3 = Fixed mass of regional LNs.

N_4 = involvement of common iliac or higher LNs.

Metastasis (M):

M_0 = No evidence of distant metastasis

M_1 = Distant metastasis.

Complications:

1- Haematuria.

2- Obstruction leading to urine retention.

3- Infection.

4- Infiltration of the surrounding structures.

- Sciatic nerve: sciatica.
- Rectum: rectal obstruction.
- Ureter: hydroureter.
- Iliac veins: DVT & oedema of both lower limbs.

5- Distant metastasis.

Incidence:

It is the commonest malignancy in males > 65 years.

Clinical picture:

Symptoms: The patient may be present with:

I- Metastatic manifestations:

5% of cases present with manifestations due to secondaries e.g. back pain or haemoptysis while the primary is silent.

II- Invasive manifestations:

Due to infiltration of the surrounding tissues e.g. sciatica, rectal obstruction,& uraemia.

III- Obstructive manifestations:

Symptoms of bladder neck obstruction as in BPH but with the following differences:

1- Short duration and the onset in order age.

2- Difficulty is marked and precedes frequency.

3- Retention is more common.

4- Haematuria is rare.

5- Pain in the suprapubic region & perineum.

IV- Complications:

Patient may firstly present with local complication e.g. retention and haematuria

Signs:

General;

Signs of the distant metastasis.

Local: = PR. examination.

The prostate is felt with the following characters:

- Asymmetrically enlargement.
- Stony hard inconsistency,
- Irregular surface
- Obliteration of posterior sulci
- The rectal wall may be fixed to the tumour.
- A sound in the urethra isn't felt

In addition, enlarged hard pelvic LNs may be plapated.

Investigations:

I- Investigations to prove the diagnosis:

1- Urine analysis: RBCs, pus cells, and malignant cells by cytology.

2- Kidney function tests: S. urea & S. creatinine.

3- Tumour markers:

1) Serum acid phosphatase (normally 0-3 King Armstrong units).

-It is elevated in 70% of cases.

-It is also elevated in Paget's disease of bone,& other carcinomas e.g. breast & gastric cancer and after PR examination for 24 hours.

2) Serum alkaline phosphatase (normally 3-13 King Armstrong units).

- It is elevated in bone metastasis.

3) S. Prostatic specific Antigen (PSA) (normally it is 0-4 ng /ml).

-It is very helpful tumour marker.

-It is also elevated in prostatitis and BPH but not elevated after PR exam.

4- IVU: it shows. -Function of both kidneys.

-Irregular filling defect in the base of the bladder.

-Back-pressure on the kidneys & ureters.

5- Trans-rectal U/S. It is very helpful and biopsy can be taken under its guide through needle introduced through perineal or rectal route.

6- Cystoscopy:

-It is valuable to differentiate cancer from BPH.

-Transurethral biopsy can be taken but perineal route is preferred.

II- Investigations to detect metastasis:

a- Chest X ray.

b- X ray on the pelvis and vertebrae.

c- Bone scan (^{99}Tcm Diphosphonate).

Differential diagnosis:

- From BPH (see the clinical picture).
- From other causes of urine retention & haematuria. (See before).

Treatment:

Operable cases

* Radical prostatectomy

Indications:

Only for localized tumour in fit patient (only 5%).

We remove:

*The prostate & Periprostatic venous plexus.

* Seminal vesicles.

* Block dissection of the obturator , internal & external iliac LNs.

Then the bladder is anastomosed to the urethra.

Complications:

Urinary incontinence & impotence.

* Many surgeons forgive performing radical prostatectomy as the alternative palliative measures are very satisfactory and there is no significant difference in survival rate.

Inoperable case

I- Hormonal therapy:

The prostatic carcinoma is a hormone dependent tumour that needs testosterone in its growth and spread.

A- Medical:

1- Oestrogen:

- It suppresses ICSH from the pituitary so, suppressing the testicular testosterone secretion.
- It has a direct antitestosterone action.

Preparations:

a) Stilbosterole. Dose: 1mg TDS.

Side effects: 1- Feminization & gynaecomastia.

2- Predispose to DVT. & heart failure and cancer breast.

b) Honovan (Stilbosterol diphosphate):

The acid phosphatase of the prostatic carcinoma splits the drug and liberating the active moiety locally in the tumour (the primary & secondaries) thus having better action with little side effects.

2- LHRH analogues (Zoladex):

-It suppresses secretion of LH from the pituitary.

-It gives equal results to castration but it is expensive.

3- Testosterone antagonists: e.g. Cyproterone and Flutamide, They are used adjuvant to castration or LHRH analogues.

4- Aminoglutethmide: to suppress the adrenal androgens (medical adrenalectomy).

B- Surgical:

1- Castration, (Bilateral orchidectomy), It was the mainstay of treatment.

2- Bilateral adrenalectomy, but it has a high mortality.

3-Hypophysectomy: Trans-cranial or trans-sphenoidal resection or better trans-ethmodial irradiation of the pituitary by Yttrium implantation.

Value of hormonal therapy:

1- It reduces the size of the tumour thus improving the obstructive manifestations.

2- Secondaries may disappear and even pathological fractures may unite.

3- Improve the general condition of the patient.

II- Radiotherapy:

-It is indicated for advanced cases with bone metastasis.

-It is given either by external irradiation or local implantation of radioactive material.

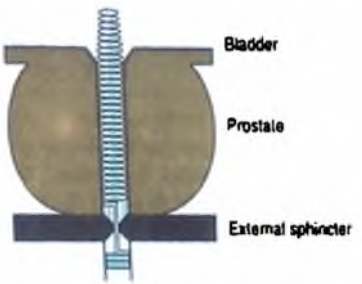
-It gives dramatic relief of pain in bone secondaries.

- Radioactive Strontium is concentrated in osteo-sclerotic secondaries and destructs them.

III- Treatment of complications e.g. urine retention:

-Trans-urethral tunneling (Laser may be used).

-Prostatic stenting is a good palliative procedure to obstructive cases.



Prostatic stenting

Prognosis:

-Radical prostatectomy cures only 50 % of operable cases but, it should be restricted to cases with a reasonable life expectancy.

-The 10 year survival rate for early tumour is 50% and for the advanced tumours is 10 %.

Symptomatology of the urinary system

1-Anuria (Suppression of urine)

Definition:

Absent urine excretion for 12 hours.

N.B: oliguria is excretion of less than 300 CC of urine in 24 hours.

Aetiology:

1- Pre-renal causes:

A- Shock All types of shock:

If systolic blood pressure drops below 70 mm Hg, the glomerular filtration ceases.

B- Crush syndrome: Due to both shock and renal shutdown by acid myohaematin.

2- Renal causes:

Due to massive bilateral renal disease e.g.:

a- Acute diffuse membranous glomerulonephritis.

b- Acute massive necrotizing papillitis.

3- Post-renal causes: -

Due to complete obstruction of both ureters or obstruction in the only functioning kidney.

Causes:

1- Calcular anuria.

2- Obstruction of both ureters by retroperitoneal fibrosis.

3- Accidental ligation of both ureters e.g. during hysterectomy.

4- Infiltration of both ureters by malignancy e.g. cancer cervix.

Calcular anuria

Aetiology:

1-Bilateral obstruction of ureters by stones.

2-Unilateral obstruction of solitary kidney.

Incidence:

It is more common in middle age males.

Clinical picture:

History: of urinary stone may be positive.

Symptoms: the patient passes through three stages:

1-Painful stage or stage of onset:

-The patient complains of severe renal colic with progressive oliguria & haematuria.

-The renal angle is markedly tender.

2-*Tolerance stage:*

- The patient feels better as the colicky pain stops but:
  - There is still anuria.
  - Rising blood urea which may reach over 200 mg %.
- This stage lasts about 3-8 days.

3-*Stage of uraemia:*

The patient begins to complain of headache, drowsiness, lassitude, nausea, hicough & constipation. Lastly, there is subnormal temperature, irregular respiration, muscle twitches and the patient passes into coma & death.

Examination:

- First, exclude retention by percussion of bladder & catheterization. It usually delivers only few drops of smoky urine.
- Palpation of the kidney:
  - The obstructed kidney is tender.
  - Recently obstructed kidney is never enlarged.

Investigations:

1-*Plain Xray*: It rarely shows the calculi, as they are usually small.

2-*Renal isotope* scan: to show the obstructed side in unilateral cases.

3-*Cystoscopy*: - It may show stone in the intramural part.

- Passing a ureteric catheter may succeed to dislodge the stone.

4-*Kidney function tests & bl. Electrolytes*: elevated S. urea & creatinine.

5-*Mannitol test*: IV mannitol fails to induce diuresis.

Differential Diagnosis:

From urine retention: The patient has intense desire for micturation, the bladder is full, and the catheter will solve the problem.

Treatment:

I-Cystoscopic catheterization of both ureters: if this succeeded to bypass the obstruction or dislodge the stone, the emergency is over & the catheters are left in place 2-3 days until general condition of the patient and function of the kidneys improve, and then treat the cause

II-Nephrostomy:

- If catheterization fails, bilateral nephrostomy is done to drain the kidneys and when the general condition & renal functions improve, treat the cause.
- until the function of kidneys improves, manage as acute renal failure.

2- Haematuria

Definition:

Passage of urine containing blood. It varies from microscopic haematuria to frank haematuria.

Aetiology:

I- General causes:

Bleeding tendency, and hypertension.

II- Local causes:

Almost all diseases of the urinary system may cause haematuria.

A- Kidney:

1-Congenital: polycystic kidney.

2-Traumatic: 95% of renal trauma present with haematuria.

3-Inflammatory: -Acute glomerulonephritis. -Acute pyelonephritis.
-Necrotizing papillitis. -Renal T.B.

4-Lithiasis: renal stones.

5-Vascular: renal infarction.

6-Neoplastic: all neoplasms of the kidney.

B- Ureters:

1-Traumatic: instrumentation.

2-Lithiasis: stone ureter.

3-Neoplastic: e.g. villous papilloma.

C- Bladder:

1-Traumatic: contusion.

2-Inflammatory: Bilharzial, tuberculous & nonspecific cystitis.

3-Lithiasis: stone bladder.

4-Neoplastic: all neoplasms of the bladder.

D- Prostatic causes:

1-Inflammation: Bilharzial, tuberculous & non-specific prostatitis.

2-Neoplastic: senile enlargement of prostate & cancer prostate. .

E- Urethra:

1-Trauma: rupture urethra.

2-Inflammation: urethritis.

3-Lithiasis: stone urethra.

4-Neoplastic: tumors e.g. papilloma.

III- False Haematuria:

- Red urine due to Ingestion of beetroots (بنجر) or drugs e .g Rifampicin & carmurate.
- haemoglobinuria.

Diagnosis

I-Clinical.

A-*History:*

1-*Age of patient:*

A- Infancy: congenital diseases, Wilm's tumour, & stone bladder.

B- Adult: Bilharzial, T.B, non-specific inflammation, and stones.

C-Old age: SEP & neoplastic diseases e.g.hypernephroma & papillary carcinoma.

2- *Types & time of haematuria:*

a- *Initial haematuria* at the beginning of micturition → urethral causes.

b- *Terminal haematuria*: At the end of micturition →bladder (trigone) causes.

c- *Total haematuria*: whole stream →kidney, ureter or bladder causes.

3-*Colour of urine:*

a- Red and contains blood clots → profuse haematuria.

*worm like clots denote supravesical causes. Discoid clots denote vesical causes.

b- Red and contains no clots → mild in alkaline urine.

c- Smoky urine → mild in acidic urine.

d- Frank haematuria (almost whole blood) →severe haematuria.

4-*Other symptoms:*

1-Pain: - colicky with stone but it may be clot colic.

dull aching due to inflammation or back pressure .

2-Frequency:

-diurnal →stone bladder. -nocturnal → S.E.P. -diurnal & nocturnal → cystitis .

3-Difficulty: - urethral causes.

4-Mass: renal or supra pubic.

5-*Past history of:*

1- Trauma & operation.

2- Drugs: e.g. rifampicin ,& anticoagulants .

3- Passage of stone.

4- Attacks of retention.

B-Examination:

1-*General examination:*

a) Pallor & hypotension → severe haematuria.

b) For general causes e.g. hypertension, & bleeding tendency.

c) General manifestations of the cause e .g. uraemia,& metastasis.

2-Local:

a) Renal swellings: - renal causes or hydronephrosis due to lower obstruction.

b) Tenderness in renal angle: renal infection or ureteric obstruction.

c) Distended bladder with chronic retention e.g. SEP.

d) Suprapubic mass in late cancer bladder.

e) PR examination, for SEP., cancer bladder or prostate.

II- Investigations:

1-Three glass test: Voiding urine in 3 glasses and compare the colour of urine in them to detect the type of haematuria. Initial (in the 1st glass), terminal (in the 3rd one) & total (in all glasses).

2-Urine analysis: for RBCs (microscopic haematuria), ova, crystals & debris.

3-Plain X-ray: for urinary calculi, calcified bladder or T.B lesion.

4-Kidney function tests.

5-Ultrasonography: for renal & UB masses.

6- IVU or ascending cystography and renal angiography.

7-Cystoscopy & pyeloscopy: the most diagnostic & can take biopsy from any suspicious lesion.

Treatment:

I-General measures:

For shocked patient, IV. Fluids, bl. transfusion, coagulants. etc.

II-Definitive ttt of the cause.

3- Retention of urine

Definition:

It is inability to evacuate a full bladder.

Aetiology:

I-Organic = Mechanical = obstructive:

A. In the lumen: Stone bladder or urethera.

B. In the wall: 1- Bladder: BNO & tumours.

2- Prostate: SEP, prostatic abscess, & carcinoma.

3- Urethra: rupture, urethritis & stricture .

C. From outside: retroverted gravid uterus, large cervical fibroid, & tumours.

II- Functional = Neurogenic:

A- Spastic (sphincter) as in:

Postoperative retention especially perineal, Gynecological operation & hysterical retention.

B- Atonic (bladder) as in:

Senility, spinal cord lesions, tabes dorsalis, drugs (atropine) & neurogenic bladder.

Pathology:

Types:

1-Acute retention: acute onset, complete retention & painful.

2-Chronic retention: Gradual onset, may be with over flow & painless.

3- Acute on top of chronic.

A- Acute retention

Definition:

Sudden failure to pass urine although the bladder is full.

Clinical picture:

1-Severe desire to micturate, but he cannot do so.

2-Severe suprapubic bursting pain, which may be periodic.

3-Full bladder on percussion, but the bladder is moderately enlarged.

Treatment:

1-Hospitalization.

2-Conservative measures:

a- Sedation with morphia.

b- Hot fomentation on suprapubic region.

c- Open a tap of water infront of the patient (psychological help).

d- allow him to sit better on the pan.

3- Medications: Parasympathomiemetics e.g. prostigmine

-Not in obstructed case (may cause rupture of bladder), as it stimulates muscle contraction.

4- Catheterization:

-Only if the above measures failed, as it may injure urethra or introduce infection. Begin with Foley's catheter, if it failed, try with Nelaton's catheter. If failed, try with metal catheter.

- If any succeeded, leave it in place and investigate the patient for the cause.

5- Suprapubic cystostomy or percutaneous suprapubic puncture: If catheterization failed.

"In all cases, you should investigate for the cause before discharge from hospital".

Chronic retention

Definition:

It is gradual distention of the bladder with increasing residual urine.

Aetiology:

It is due to progressive atony of detrusor muscle 2ry to:

1-Obstruction: -SEP - Cancer prostate.

- BNO - stricture urethra.

2- Neurogenic: Spinal cord lesions.

Clinical Picture:

- History of the cause is usually evident.
- The patient may present early with marked frequency, and late with retention & overflow. When the distention of the bladder is maximum, each drop of urine comes out from the ureters another drop escapes from the urethra (false incontinence).
- The patient is unaware of bladder distention, as it is painless.
- Abd. exam. reveals markedly distended non-tender bladder that may reach above umbilicus.
- PR. examination may reveal the cause e.g. SEP. or cancer prostate.

Treatment:

- Hospitalization:
- Before any attempt to deflate the bladder, the bl. urea should be estimated.
  * If below 100 mg %, deflate the bladder and investigate for he cause.
  *If > 100mg%, slow (Tidal) decompression ,as sudden deflation leads to reactive hyperaemia in the kidney and may lead to profuse haematuria, acute tubular necrosis & renal failure

"In all cases, you should investigate for the cause before discharge from hospital." .

4 –Swelling

Renal swelling may be due to:

I- Congenital: - Polycystic kidney, & solitary renal cyst.

II- Traumatic: - subcapsular haematoma, & pseudo haemato hydronephrosis.

II- Inflammatory: - Secondary pyonephrosis, & perinephric abscess and renal carbuncle.

IV- obstructive: Hydronephrosis.

V -Neoplastic: - Hypernephroma, & Wilm's tumour.

Renal swelling has the following characters:

1- Intra-abdominal swellings in the hypochondrium and encroach on lumber & iliac regions.

3- It either fills the loin or can be pushed to it.

4- It moves up & down with respiration unless fixed.

5- It usually maintains its reniform shape with rounded borders.

6- The hand can be insinuated under the costal margin to get its upper pole.

7- It shows the renal ballotment unless fixed.

8- It is dull on percussion with band of resonance over it (the colon).

- Clinical findings in common renal swelling

Item	Polycystic kidney	Solitary renal cyst	Secondary pyonephrosis	Hydro-nephrosis	Wilm's tumour	Renal cell carcinoma
I-General	-Rickets -hypertension -uraemia		-Toxaemia	-Uraemia	-Hypertension -Fever -Secondaries	-Secondaries -Hypertension -Fever
II-local						
-Size	Mild	Mild	Moderate	Moderate	Huge	Mild
-Shape	Reniform	Globular	Reniform	Reniform	Irregular	Irregular
-Surface	Fine lobular	Smooth	Lobular	Lobular	Irregular	Irregular
-Tenderness	No	No	Tender	No	No	May be tender
-Consistency	Firm	Cystic	Cystic	Cystic	Hard	Hard
-Mobility & ballottement	freely mobile	mobile	Mobile	Mobile	Usually fixed	Early mobile Late fixed
III- PR exam	free	Free	Predisposing. factor	Cause eg SEP	Free	Free

5- Frequency

Definition:

Frequent micturition either due to increased amount of urine secretion or due to bladder cause.

Aetiology:

I- Polyuria: (false frequency):

Increased urine secretion (see causes in internal medicine).

II- True frequency:

The amount passed with each voiding is less than the normal bladder capacity (about 300 – 400 cc).

A- Irritation of the bladder: e.g. cystitis, stone & crystaluria.

B- Decreased bladder capacity e.g. SEP, contracted bladder, external compression on the bladder by pregnant uterus.

C- Neurological diseases & hysterical frequency.

Type of frequency:

1-Nocturnal frequency: as in SEP.

2-Diurnal frequency: as in stone bladder.

3-Diurnal & nocturnal frequency: as in cystitis.

6 – Difficulty

Definition:

Difficulty in voiding urine.

Actiology:

1-S E P. 2-B N O. 3-stricture urethra.

In SEP. the stream is not improved, but even worsened by straining, while difficulty improved with straining in BNO & stricture urethra.

7- Dysuria

Definition:

Pain or discomfort in voiding urine.

8- Strangury (Bladder tenesmus)

Definition:

It is intense desire to micturate with passage of few drops of urine (sense of incomplete evacuation). It is due to cystitis

9 – Urgency

Definition:

Urgent desire for micturation that may overcome the normal control of sphincter.

10- Pyuria

Definition:

Passage of turbid urine containing pus cells due to infection elsewhere in the urinary tract:

11- Crystaluria & Lithuria

Definition:

They mean passage of crystals or stones with urine.

12- Necroturia

Definition:

Passage of necrotic tissues in urine. It is highly suggestive of bladder cancer.

Chapter XI

The scrotum

The testes, Epididymis & spermatic cord

Embryology

-The testicle develops in the abdomen from a mesodermal ridge **(Genital ridge)** on the back of the coelomic cavity. The testis develops from the middle part of this genital ridge in close relation to the middle group of ***mesonephric tubules***, which will be utilized by the testis to form the vasa efferentia.

-The cranial part of the genital ridge will disappear, while the caudal part will form ***the gubernaculum*** which connects the lower pole of the testis to ***the genital swelling*** (the future scrotum), and plays an important role in the descent of the testis.

-The testes begin to descend in the posterior abdominal wall reaching the deep ring in the 7th intrauterine month. Passing through the inguinal canal, it reaches the superficial inguinal ring in the 8th month and reaches the scrotum in the 9th month.

- During descent of the testis, It carries with it a fold of peritoneum called ***the processus vaginalis*** which will supply the testes with a serous covering i.e tunica vaginalis and then obliterates forming the vestige of processus vaginalis.

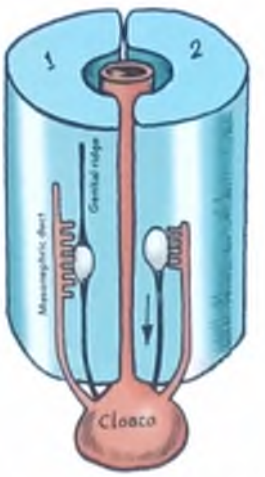
- Factors favoring descent of the testis.

1- Elongation of the body of the embryo.

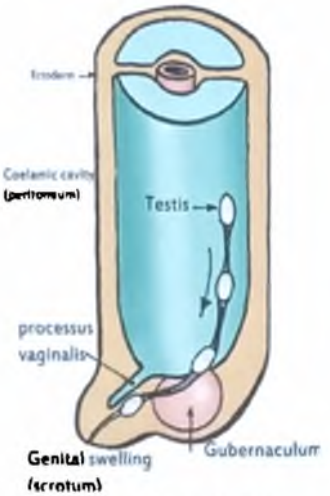
2- Contraction of the gubernaculum pulls the testes towards the scrotum.

3- The hormonal factors: the maternal HCG reaches the foetus → growth and engorgement of the testes that become heavier and so, descends.

4- The intra-abdominal pressure squeezes the testes to the scrotum.



Development of the testes



Descent of the testes

Anatomy

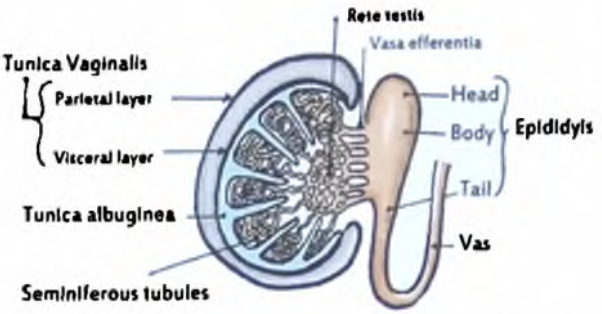
-Each testis is about 2 x 1 x 1 inch.

- It has a true capsule called tunica albuginea which is formed of tough fibrous tissue .

- It is surrounded with two layers of tunica vaginalis namely the visceral & parietal layers.

-It has two borders (ant. & post.), two surfaces (medial & lateral), & two poles (upper & lower).

-It is divided into numerous compartments. Each compartment contains ***semineferous tubules,*** which anastomose together near the posterior border of the testis (mediastinum testis) forming what is called ***rete testis*** from which, about 15-20 ***vasa efferentia*** emrge outside the testis to join ***the epididymis.*** The later is formed of head, body & tail. The head of epididymis is related to the upper pole of the testis, the body is posterolateral to the testis . At the tail of the epididymis ***the vas deferus*** begins and ascends on the medial surface of the testis



Internal anatomy of the testis

Blood supply:

I-Arterial: the testis gets its blood supply from:

1-Testicular artery: It arises from the abdominal aorta at the level of L_2

2-Cremasteric artery: from the inferior epigastric artery.

3-Artery of the vas: from the inferior vesical artery.

4-Scrotal arteries from the internal pudendal artery

II-Venous drainage : the testis drains into :

1-Pampiniform plexus, which become 4 to 6 veins in the inguinal canal then, become 1 to 2 testicular veins that drain into IVC on the right side and Lt. renal vein on the left side.

2-Cremasteric veins: They anastomose with the pampiniform plexuses and drain into the inf. epigastric veins.

Nerve supply:

Sympathetic fibers from T_{10} . T_{12} run with testicular arteries to the testis.

Lymphatic drainage:

Along the testicular artery to end in para-aortic LNs around the origin of the testicular artery of both sides. Some lymphatics may pass with the lymphatics of the vas to the internal iliac LNs.

Layers of the scrotum

1-Skin.

2-Dartos muscle in the superficial fascia

3-External spermatic fascia.

4-Cremastric muscle and its fascia.

5-Internal spermatic fascia.

6-The parietal layer of tunica vaginalis.

7-The visceral layer of the tunica vaginalis.

I- Congenital Anomalies

1-Maldescended testis

1-Arrested (undescended) testis

Definition:

Arrest of the testicular descent at any point in its normal pathway to the scrotum.

Aetiology:

1-Deficiency of gonadotrophins.

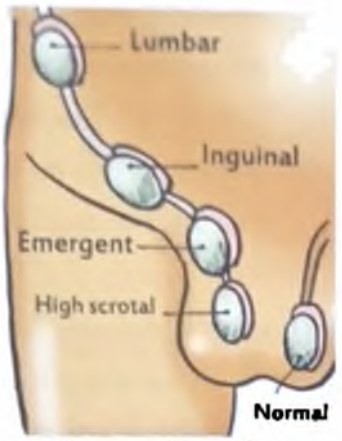
2-Abnormality of the gubernaculum.

3-Anatomical abnormalities that hinder the descent of the testis.

a) Adhesions: retroperitoneal or in the inguinal canal.

b) Short testicular vessels.

c) Narrow inguinal rings.



Arrested testis

Pathology:

Site : The testis may arrest any where along the normal pathway :

1- Intra–abdominal testis (in the abdomen).

2- Inguinal testis (in the inguinal canal).

3- Subinguinal or high scrotal testis (at the neck of the scrotum).

Up to six months, the testis remains almost normal. After that, there is retardation of growth due to the higher temperature in the abdomen. If it was left until puberty, permanent damage to the germinal epithelium takes place.

Complications:

The arrested testis is liable to:

1-Atrophy: in neglected cases, atrophy of the testes with failure of spermatogenesis occurs. However, the interstitial cells, responsible for hormone secretion are not affected.

2-Associated interstitial hernia (in 80% of cases) which may strangulate.

3-Psychological troubles and sense of sexual inferiority.

4-Torsion: 2% of arrested testes undergo torsion.

5-Trauma: arrested tested are more liable to trauma.

6-Tumour: all types of malignant tumours of the testis are commoner in the arrested testis and the higher the position of the arrested testis, the commoner is malignancy. The tendency for malignancy is not abolished by surgical correction .

Incidence:

-It occurs in 4% of full term babies and in 30% of premature.

-It is commoner on the right side due to later descent of the right testis, bilateral in 20% of cases.

Clinical picture:

Symptoms:

- One side of the scrotum is empty

-The patient may present due to failure in medical commission.

-The patient may present with complications

Signs:

-One or both scrotal compartments are empty & underdeveloped.

-Inguinal & subinguinal testes can be felt in their places.

Investigations:

1-Ultrasonography & MRI: to localize the site of impalpable testis.

2-Hormonal assay (LH & testosterone) to exclude anorchism in bilateral cases.

3-Laparoscopy is very helpful in lumbar testes.

Differential Diagnosis:

I-Subinguinal testis from retractile testis. In retractile testis:

1-There is well-developed scrotum with rugae.

2-The testis can be pulled to the buttom of the scrotum.

3-Chair test is helpful (see later).

II-Inguinal arrested testis from inguinal ectopic one:

Inguinal arrested testis	Inguinal ectopic testis
-the cord is lateral to the testis.	The cord is medial to the testis.
-the testis can be pushed laterally but not medially	The testis can be pushed medially but not laterally .
-The testis becomes less apparent on straining.	The testis becomes more apparent on straining (SC).

Treatment :

I-Medical (Hormonal) treatment: *" it is mostly a test rather than remedy"*

Indicated only in:

1. bilateral cases with suspected hypogonadism.
2. preoperatively to increase the size of the testis.
3. Chorionic gonadotrophin 100 IU. /Kg twice weekly for 5 weeks.

II- Surgical treatment: dartos pouch orchidopexy:

Time: before 6 months old to avoid atrophy of the testis.

Procedure:

1-The testis and the cord are mobilized.

2- Herniotomy of the associated hernia.

3- The testis is placed in the scrotum in dartos pouch. where it is secured by non-absorbable sutures

Dartos pouch orchidopexy

Treatment of Problematic cases

I- impalpable undescended testis:

Laparoscopy is the first line for diagnosis and treatment. The vas and the testicular vessels are followed throughout their courses. There are a number of possible findings:

- Blind-ending vas and vessels with no evident testis: the so-called 'vanishing testis → nothing done
- Normal or attenuated vas and vessels entering through the internal ring: → open inguinal exploration and orchidopexy of normal testis or orchidectomy of atrophic one
- Normal size testis but high in retroperitoneum (short pedicle) → dissection to bring it in inguinal canal then trial of one stage or two stages orchidopexy

***Fowler Stephens operation**: (two stage orchidopexy)

- First stage: high division ligation of the testicular vessels.
- The testis is left in its high position to attain new collaterals from rest of vessels (cremasteric and vassal vessels)
- Second stage: after 6 months, the testis is mobilized on its new vessels and orchidopexy is completed.

***Microvascular technique**: Division of the testicular artery, which is re-anastomosed to the inf. epig. artery.

II-Neglected atrophied cases:

A- Unilateral → orchidectomy to prevent hazard of malignancy.

B- Bilateral cases: Do right orchidectomy and leave the left for its endocrine function We remove the right as torsion in right arrested testis simulates acute appendicitis and usually misdiagnosed.

2-Ectopic testis

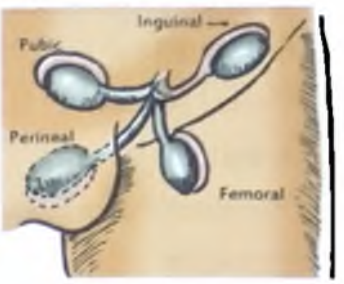
(Deviated testis)

Definition:

Testis lies in a place outside its normal pathway of descent.

Aetiology: unknown:

It may be due to rupture of the scrotal tail of the gubernaculum and the testis is pulled by one of its accessory tails, which are inguinal, femoral, pubic, or perineal .



Ectopic testis

Pathology:

Sites = Types :

1-Inguinal ectopic testis: superficial to the inguinal canal.

2-Perineal ectopic testis: in the superficial perineal pouch.

3-Femoral ectopic testis: in the femoral triangle.

4-Pubic or pubo-penile ectopic testis: over the root of the penis.

As the testis lies in the subcutaneous tissue, the spermatogenesis is not affected.

Complications:

Like arrested testis except for atrophy & sterility.

Treatment:

Orchidopexy, which is much easier than in arrested testes as the vessels & vas are long enough to deliver the testis to the bottom of he scrotum.

3-Retractile testis

Aetiology:

In most children, the testes are hypermobile due to their low weight and over activity of the cremaster muscles.

Chair test

Clinical picture:

-Stimulations like fear, medical examination, or palpation with cold hand stimulate the cremaster to draw the testis up to the inguinal canal.

-Reassurance & gentle exam. with warmed hand help to pull the testis to the bottom of the scrotum.

-In difficult cases. ***"chair test"*** is done by putting the child on a chair and his knees against his chest or putting him in squatting position.This helps to squeeze the testis to the scrotum.

Treatment:

It requires no treatment. Only reassurance of the parents because after puberty the testis will stay in the scrotum as it will be too heavy to be pulled by the cremaster muscle.

II-Epididydmal cyst & Spermatocele

Aetiology: It is either:

1-Dilatation of mesonephric remnants.

2-Retention cyst of the epididymis secondary to obstruction of vasa efferentia.

Pathology:

Unilocular cyst related to the head of the epididymis which may contain clear fluid (epididymal cyst) or whitish fluid that contains dead & living spermatozoa (spermatocele).

Clinical picture:

Symptoms:

It is usually asymptomatic, discovered accidentally during examination for unrelated condition.

- It may be so large that the patient imagines that he has three testicles.

Sign:

- The cyst is felt as small soft cystic swelling with smooth surface related to but inseparable from the upper pole of the testis.
- Epididymal cyst is translucent while spermatocele is opaque in transillumination.

Differential diagnosis:

From other cysts in the scrotum especially encysted hydrocele of the cord, (see later).

Treatment:

Small asymptomatic cyst: no treatment

Large cysts: excision.

III- Inversion of the testis.

Definition:

Abnormal position of the testis inside scrotum.

Types:

1-Anterior inversion: The epididymis lies anterior to the testis.

2-Polar inversion: either complete when the upper pole of the testis & head of the epididymis lies inferiorly or incomplete when the testis lies horizontally in the scrotum (transverse lie).

II-Trauma

I- Torsion of the testis.

Definition:

It is a sudden twisting of the testis and spermatic cord causing strangulation of blood vessels and even infarction of the testis.

Aetiology:

Predisposing factors:

1- *Inversion of the testis*. All types of inversion are the commonest predisposing factors.

2- *Arrested & ectopic testis.*

3- *Wide high investment of the tunica* makes the testis hang in the tunica like a clapper in a bell.

Precipitating factors:

-It is usually precipitated by strain e.g straining at stool, lifting heavy objects or during intercourse.or even crossing the legs.

- It is thought that the spirally arranged cremaster muscle begins the torsion and together with the pulsation of the twisted testicular artery will complete and maintain it.

Pathology:

- The lower pole of the testis rotates in a direction away from the midline (septum dartos) i.e. clockwise on the right and anticlockwise on the left side.
- The testis becomes congested & oedematous.

Complications:

1- Infarction of the testis if torsion is > 360 degrees or > 24 hours.

2- Sterility: if an infarcted testis is neglected. In this case, the blood-sperm barrier is breached and immune cells may recognize sperms as foreign antigen and begin to form antibodies against them, which reach the other normal testis causing sterility.

Torsion of the testes

3- Secondary hydrocele.

4- Shock (neurogenic) and paralytic ileus.

Incidence:

It has two peaks of incidence:

1- Adolescents (10-25 years) (the commonest)

2- Infancy (the 2nd common).

Clinical picture:

Symptoms:

1- Sudden severe pain in the groin and abdomen.

2- Vomiting at the onset.

3- Abdominal distention and may be collapse.

Signs:

I- Torsion of a completely descended testis.

- The testis is felt as tense very tender swelling at the neck of the scrotum.
- The scrotum is red & oedematous.
- Twists may be felt in the spermatic cord.

II- Torsion of an arrested testis:

There is tender inguinal swelling that simulates strangulated inguinal hernia or acute appendicitis on the right side, but characteristically, the scrotum is empty.

Investigations:

Duplex ultrasound will show the twist and thrombosis of the testicular vessels.

Differential diagnosis:

I- From acute epididymo-orchitis. The onset is less dramatic, the patient is feverish, the testis is in its place, with no twist of the cord. In addition, urine analysis reveals pus cells.

II- From strangulated inguinal hernia & acute appendicitis. The testis is felt in its normal place.

Treatment:

Time factor is very important. Exploration is mandatory if suspicion develops and without waiting for investigations. A misdiagnosis of testicular torsion leading to an unnecessary 'scrotal exploration' does not lead to medical litigation (مساءلة طبية).

- Immediate exploration through inguino-scrotal incision.
- Undo the twist of the cord and examine the testis.
  * If viable, evert the tunica and fix the testis to the bottom of the scrotum to avoid recurrence.
  * If not viable, resect it.
- In both conditions, we should fix the other testis in the same way because torsion is common to recur on the contralateral side.

II- Traumatic Hydrocele & haematocele

(see later).

III- Inflammatory conditions

Acute inflammation

1- Acute epididymo-orchitis.

Aetiology:

I- Causative organisms:

A- *Bacterial*: Gm +ve: Staph. & Strept.

Gm-ve : gonococi (not common nowadays) , E.coli (the commonest) & proteus .

B- *Viral*: mumps.

II- Route of spread.

1- Ascending infection (common) from urethra, prostate & seminal vesicles.

2- Haematogenous (rare) as in mumps.

III-Predisposing factors:

Instrumentation or operation on bladder or prostate.

Pathology:

The inflammation involves the testis & epididymis with marked oedema & congestion.

Complications:

1- Obstruction: of the epididymis. Sterility may result in bilateral cases.

2- Testicular atrophy or sloughing.

3- Secondary hydrocele & pyocele.

4- Residual mass in the tail of the epididymis (firm & tender).

Clinical picture:

General:

Constitutional manifestations (fever, headache, anorexia, & malaise).

Local:

- The scrotum is red, swollen & worm.
- Marked pain and tenderness over the testis & epididymis.
- Secondary hydrocele is common.
- Picture of complications.
- PR examination, may elicit tenderness in the prostate & seminal vesicles in ascending type.

Differential diagnosis:

From other painful tender inguino-scrotal swellings e.g.strangulated inguinal hernia & torsion testis.

Treatment:

General: Rest, antibiotics, analgesics, & antipyretics.

Local: - Elevation of the scrotum, & hot fomentations.

-Drainage of pyocele or residual abscess.

Chronic inflammations

1- Chronic non-specific epididymitis.

2- Tuberculous epididymitis.

3- Bilharzial funiculo-epididymitis.

4-Filarial Funiculo epididmyitis.

5- Syphilitic orchitis.

6- Lepromatous orchitis.

Inflammation of the scrotum

Non specific inflammation:

Abscess, furuncles & cellulitis.

Specific inflammation:

- Erysipelas, TB. Sinuses (posterior), syphilitic sinus (anterior).

Idiopathic gangrene of the scrotum

Fournier's gangrene

Definition:

It is an infective gangrene of the scrotum.

Aetiology:

I-Causative organisms: mixed infection, microaerophilic strept., staph., & Cl. Welchii.

II- Route of infection: direct contamination of perineal wound or drainage of periurethral abscess.

III- Predisposing factors: It usually occurs in immunocompromized persons e.g. diabetics.

Clinical picture:

Early, there is inflammation & constitutional manifestations, then gangrene of the skin with line of demarcation & toxaemia.

Later, the skin of the scrotum sloughs leaving the testis hanging, bared, but healthy.

Fournier's gangrene

Treatment:

Medical: Antibiotics (cephralosporine + aminoglycosides) + control of diabetes.

Surgery: Removal of gangrenous skin when line of separation appears, then repeated dressing and grafting or plastic reconstruction.

IV- Varicocele

Definition:

It is dilatation, elongation & tortuousity of the pampiniform plexus of veins.

Aetiology:

I- Primary varicocele: unknown, but may be due to:

1- Congenital weakness of veins.

1- Congenital absence of valves normally present at the termination of the testicular veins.

Predisposing factors:

1- Prolonged sitting or standing.

2- Chronic constipation.

3- Congestion due to unrelieved sexual excitement.

II- Secondary varicocele:

Due to obstruction of the testicular veins by abdominal or pelvic masses e.g. hypernephroma.

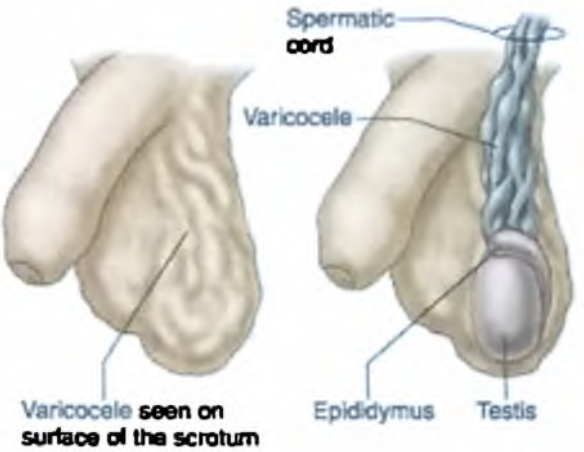
Pathology:

I- Primary varicocele:

- The common variety.
- It affects young adults (15-30 years).
- The veins are patent but incompetent.

II- Secondary varicocele:

- It is rare.
- It affects old age.
- The veins are obstructed in the abdomen.



Complications:

1-Hypofertility & infertility especially in bilateral cases due to:

a) Increased temperature in the scrotum secondary to venous congestion inhibits spermatogensis.

b) Reflux of steroids & catecholamine, which are spermatogensis inhibitors, from left renal vein to the Lt. testicular vein in a toxic concentration to both testes. This results in suppression of spermatogensis in both testes.

2- Tesricular atrophy: in long standing cases.

3- Secondary hydrocele.

4- Thrombophlebitis, which may end in spontaneous cure.

Incidence:

- Primary varicocele is common in young males (15-30 years), while secondary varicocele is common in middle & old age (> 40 year).
- It is common on the Lt. side due to :
  - ❶ The Lt. testis usually hangs lower than the right one, thus the Lt. testicular vein is longer.
  - ❷ The Lt. testicular vein opens at a right angle into the Lt. renal vein, while the right one opens obliquely into the IVC.
  - ❸ The sigmoid colon rests and may compress on the Lt. testicular vein.
  - ❹ The Lt. renal vein may be compressed in the angle between the aorta and the superior mesenteric artery (the nutcracker mechanism).

Clinical picture:

Symptoms:.

I- Asymptomatic cases: Most cases are asymptomatic, and discovered accidentally during routine examination or failure of medical commission.

II- Symptomatic cases::

1- *Pain*, which may be:

- Dull aching pain in the testis due to congestion.
- Dragging pain in the cord in late cases increased by prolonged standing & hot weather.

2- *Swelling:* In moderate & severe cases, the patient feels the scrotum as a bag of worms.

III- Cases may present with complications: e.g. thrombosis, & infertility.

Signs :

I-General:

- Examine the abdomen, pelvis & neck to exclude masses in secondary varicocele.
- Search for other associated conditions as piles, VV. & hernia.

II Local:

-Inspection:

* The affected testis usually hangs lower than normal.
* The scrotum is redundant with visible veins.
* The varicose veins may be visible under the skin of the scrotum.

-Palpation:

* In standing position, it is felt as multiple, soft cystic, compressible inguinoscrotal swellings.
* There are expansile impulses and even thrill on cough.
* The swellings disappear on lying down and elevation of the scrotum (only in 1ry varicocele)

* Signs of complication e.g. atrophied testis & secondary hydrocele.

Investigations:

1- Scrotal duplex: to confirm the diagnosis.

2- Abdomino-pelvic U/S to exclude masses & 2ry varicocele.

3- Semen analysis, to detect oligospermia.

Differential diagnosis:

I- From other inguino-scrotal swellings (see before).

II- Primary from secondary varicocele.

	Primary varicocele	Secondary varicocele
Patient age	15-30 years	Usually above 40 years
Onset & duration	Gradual onset & long duration	Acute onset & short duration
Side	Usually left side	Left or right
Change with posture	Disappears on lying down	No change with posture
Abdominal exam	Free	Pelvic or abdominal swelling

Treatment:

I- Conservative ttt:

Indications .In most cases (mild noncomplicated).

It includes:

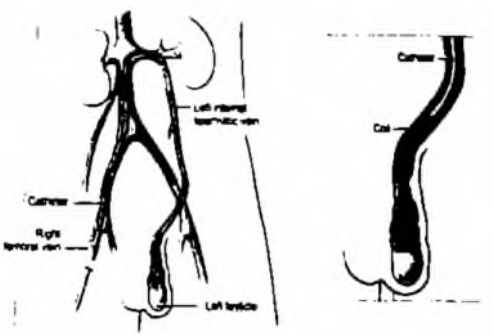
- Avoid predisposing factors e.g. prolonged standing, straining & sexual excitation.
- Frequent cold baths.
- Scrotal suspender may relieve the pain but increases warmth and decreases fertility.

II- Injection sclerotherapy: It is still under trial.

III- endovascular embolization of the main veins

III- Surgery.

Indications: 1- Cases complicated with infertility.
2- Large painful varicocele that interferes with physical activity.



Endovascular embolization

Procedures:

A- Scrotal approach:

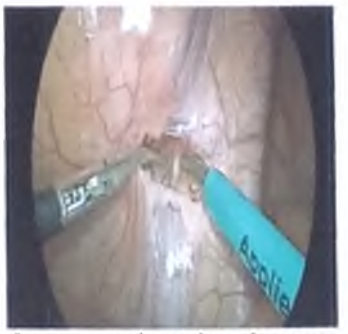
1- *Multiple ligatures (delta operation):* The anterior of the pampiniform plexus are ligated continuity at points of their junction. The tunica vaginalis is everted to prevent hydrocele.

2- *Ligature excision:* The anterior group of veins is dissected. ligated at two levels and excis The ends are transfixed and are tied together to elevate the testis and decrease congesti The tunica is also everted

B- Inguinal approach: The anterior group of veins is ligated, excised in the inguinal canal. The ends are approximated as before.

C- Abdominal (High) approach (Palomo's operation):

- High inguinal incision with muscle splitting incision.
- Testicular veins are identified in the retroperitoneal tissues. They are ligated and excised as high as possible. Now, it is the standard operation for varicocele especially in case of oligospermia. It should be bilateral procedure.



Laparoscopic varicocelectomy

D- Laparoscopic approach:

Testicular veins are identified after they emerge from the deep ring and clipped laparoscopically

V- Hydrocele

Definition:

Collection of fluid in tunica vaginalis or remnant of processus vaginalis.

1- Congenital hydrocele.

(Abdomino vaginal hydrocele)

Actiology:

Persistence of the whole processus vaginalis that remains communicating with the peritoneal cavity through a small hole. This hole is too narrow to allow herniation of abdominal viscera and transmits fluid only.

Congenital hydrocele

Clinical Picture:

Symptoms:

The mother notices a scrotal swelling in her baby, which is more marked at the end of the day.

Signs:

A cystic translucent inguinoscrotal swelling which is irreducible and shows no impulse on crying.

Differential Diagnosis:

From congenital hernia which is reducible and shows impulse on crying and resonance on percussion.

Treatment:

- Conservation can be allowed for 2 years because spontaneous resolution occurs in most cases.
- Divide the sac into two parts. The upper part is transfixed at the neck like a hernial sac .The lower part is evacuated and left open or everted around testis.

2-Infantile hydrocele

(Funiculo-vaginal hydrocele)

Infantile hydrocele

Aetiology:

Persistence of the whole processus vaginalis, but it loses its communication with the peritoneal cavity at the deep ring.

Clinical picture:

-It is common in infants, but may affect older age group.

-The swelling closely simulates congenital hydrocele, but does not show a diurnal variation in size.

Treatment:

As congenital type.

3-Encysted hydrocele of the cord

Aetiology:

Persistency of the intermediate part of the processus vaginal that loses its communication with both peritoneal cavity and the tunica vaginalis.

Encysted hydrocele of the cord

Clinical picture:

-It commonly presents in childhood & usually unilateral.

-It is oval, cystic, translucent scrotal swelling (usually at the neck of scrotum), related to the upper pole of the testis but can be easily separated from it.

-When the testis is pulled down, it becomes less mobile.

Differential diagnosis:

From other scrotal swellings, especially spermatocele, epididymal cysts. (see later)

Treatment:

Excision through scrotal or inguinal incision

4- Hydrocele of hernial sac

Aetiology:

A hernial sac, which shuts off the peritoneal cavity and fluid collects within it.

Hydrocele of hernial sac

Clinical picture:

-History of hernia is diagnostic.

-Inguinal or inguinoscrotal swelling which is pyriform, cystic, and translucent

-Traction on the testis does not affect its mobility.

Treatment:

Complete excision and repair of the inguinal canal.

5- Hydrocele of canal of Nuck

- It simulates the encysted hydrocele of the cord, but it occurs in females and is related to the round ligament of the uterus.
- It forms an inguinal or inguinolabial swelling, which is cystic and translucent.
- Treatment: Excision.

6-Vaginal Hydrocele

Aetiology:

Vaginal hydrocele

1-Primary hydrocele (commoner):

-It is of unknown aetiology .It may be due to:

1-Increased secretion or decreased absorption of fluid in the tunica.

2-Repeated minor unnoticed trauma.

3-Subclinical infection (the most accepted).

2-Secondary hydrocele:

- Hydrocele secondary to:

1-Trauma. 2-Acute epididymo–orchitis.

3- TB or syphilis. 4- Varicocele.

5- Testicular tumours. 6- Post operative: e.g. after operations on ing. hernia

Pathology:

The fluid accumulates in the tunica vaginalis (completely obliterated processus vaginalis) and has the following characters:

* Straw yellow in colour.
* Specific gravity between 1020 and 1024.
* 6% albumin content (suggestive of exudate).
* In longstanding cases, the cholesterol content rises markedly.

Types:

1- Primary hydrocele	2- Secondary hydrocele :
No obvious cause.	There is an evident cause.
It is usually large & tense.	It is usually small & lax.

Complications:

1- Rupture: spontaneous or by trauma. 2- Infections will result in pyocele.

3- Calcification of the sac. 4- Atrophy of the testis in longstanding cases.

5- Interference of daily activities and impotence in large hydrocele, which may bury the penis (mechanical impotence).

6- Hernia of the hydrocele sac: in long standing cases, the hydrocele sac may herniate through a gap in the dartos muscle.

Incidence:

It is the commonest intrascrotal swelling.

It is common in middle aged (1ry) and old age (2ry) males.

It is commonly unilateral, but may be bilateral.

Clinical picture:

Symptoms.

- Painless, slowly growing scrotal swelling.
- Symptoms of complications.

Signs:

I- Primary hydrocele: Cystic scrotal swelling which has the following characters:

* Single (unilateral or bilateral).
* Site: pure scrotal
* Size : usually large
* Shape : pyriform
* Surface : smooth
* Skin over: is free
* Surrounding structures of the cord are free
* It is brilliant translucent.

II- Secondary hydrocele:

- Signs of the cause may be evident.
- It is usually small lax cystic swelling which is detected by :

  1) Obliteration of the sulcus between the testis & epididymis.

  2) Pinching test, the parietal layer of the tunica can be pinched from the visceral layer.

Investigations:

Scrotal ultrasound to detect the cause in secondary hydrocele.

Differential Diagnosis:

From other scrotal swellings (see later).

Treatment:

I- Surgical:

1-Eversion of the tunica:

- Through scrotal incision.

- The sac is incised longitudinally and everted to be sutured behind the epididymis.
- Then the testis is replaced in the scrotum and the wound is closed with a drain.

2-Subtotal excision of the tunica:

It is indicated in -Large hydrocele.

-Thickened fibrosed or calcified hydrocele.

Lord's operation

Procedure:

- The sac is dissected and incised as before.
- Then it is excised close to its reflection on the epididymis.
- The cut edge is either sutured or everted as before.
- The testis is replaced and the wound is closed with drain.

3- Lord's operation:

It is indicated in small hydrocele in which the tunica is not thickened or calcified.

Procedure:

- Scrotal incision through all layers of the scrotum including the parietal tunica.
- The testis is allowed to prolapse through the incision so that the tunica is everted on the other layers of the scrotum.
- Then the tunica is plicated with a series of radially arranged continuous sutures from the cut edge of the tunica to its reflection at the junction between the testis and the epididymis.
- Then the testis is replaced in the scrotum and the wound is closed without drain.

Advantages:

- No dissection, so no bleeding, and no haematoma.
- No oedema and no need for drain.

II-Tapping – Aspiration

It is indicated only in unfit patients with large hydrocele as a palliative procedure.

Fluid is aspirated with small bore needle under local anaesthesia and complete aseptic conditions.

Complications

1- Infection → Pyocele.

2- Haemorrhage → haematocele.

3- Puncture of testis.

4- Recurrence in 100% of case.

N.B. Injection of sclerosant material after aspiration to lower the recurrence rate is not advised.

Criteria of secondary hydrocele due to testicular tumours on aspiration:

1- Red in colour.

2- Residual mass after aspiration.

3- Rapid recurrence.

4- Revealing malignant cells in cytological examination.

VI- Neoplasms of the testis

99% of testicular tumours are malignant Benign tumours are rare including adenoma and some interstitial cell tumours.

Actiology:

The premalignant conditions are arrested or maldescended testis and testicular dysgenesis

Pathology:

***Types = Classification:**

I- Germ cell tumors:

- * Seminimatous: - - spermatocytic tumours (rare benign tumour)
  - Seminoma (40%)
- * Non seminimatous: - Teratoma: (32 %).
  - Choriocarcinoma
  - Embryonal carcinoma
  - Yolk sac (endodermal sinus) tumor

II- Non germ cell tumors:

- *Sex cord stromal tumors: - Sertoli cell tumors
  - Leydig cell tumors
- * Lymphoma

I- Germ cell Tumours: (mostly malignant)

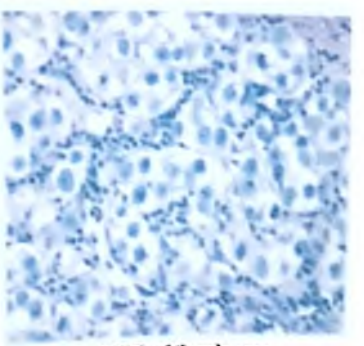
A- Seminoma (40%)

Origin: From the seminferous tubules (adenocarcinoma of the seminferous tubules).

Incidence: It is common between 30 to 50 years.

MAC: -A large firm non-capsulated mass with smooth surface.
-Cut section: is pinkish creamy homogenous, but in rapidly growing tumours, there are areas of haemorrhage & necrosis.

MIC: -The malignant cells are rounded with clear cytoplasm and large nuclei arranged in sheets separated by fibrous stroma.
- There is lymphocytic infiltration in the tumour that reflects good immune reaction against the tumour.



MIC of Seminoma

Spread: mainly by lymphatics, and rarely by blood.

Special character: highly radiosensitive.

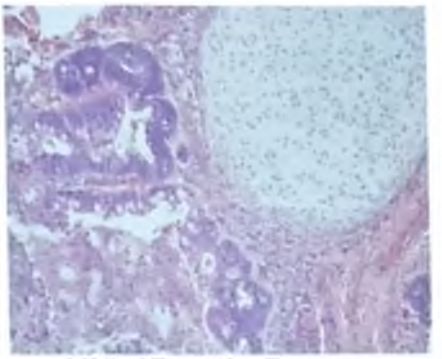
B-Teratoma:

Origin: From Totipotent cells present in the rete testis. These cells are capable to differentiate into any ectodermal, mesodermal or endodermal tissues.

Incidence: It usually occurs in young age group (10-30 years).

MAC: -Variable irregular masses which are cystic in consistency and may have hair & teeth.
-Cut section is usually yellowish with areas of hemorrhage & necrosis.

MIC: -collection of differentiated cells or organoid structures, such as neural tissue, muscle bundles, islands of cartilage & clusters of squamous epithelium.



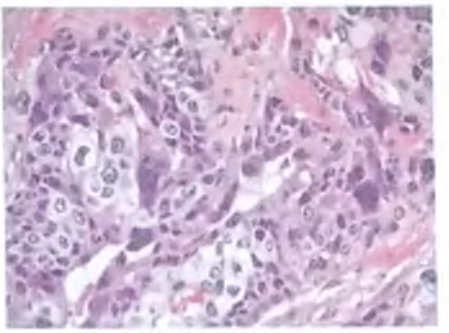
MIC of Testicular Teratoma

Spread: Teratoma spreads mainly by blood.

Special characters: It may be functioning

C- Choriocarcinoma:

- It was considered a subtype of teratoma.
- It is composed of syncytial cell mass with malignant villous or papillary cytotrophoblast.
- It is the most malignant tumour known with blood metastases even before a clinical mass .
- It commonly secrets gonadotrophin.



MIC of Choriocarcinoma

D- Embryonal Carcinoma:

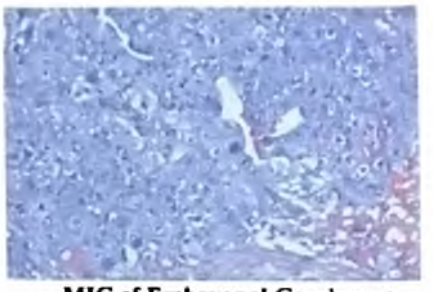
Origin: primitive germ cells.

Incidence: It occurs mostly in the 20- to 30-year

MAC: - smaller than seminoma and do not replace the entire testis.
- Cut section is heterogeneous with areas of necrosis and haemorrhage
- infiltration through the tunica albuginea into the epididymis or cord frequently occurs

MIC: The tumor is composed of solid and glandular proliferation of cells with pale cytoplasm and enlarged, highly pleomorphic nuclei which are pale & irregular.

Spread: early spread to lymphatics and blood



MIC of Embryonal Carcinoma

Special character : more aggressive than seminomas.

E- Yolk Sac Tumor

Origin: primitive germ cells

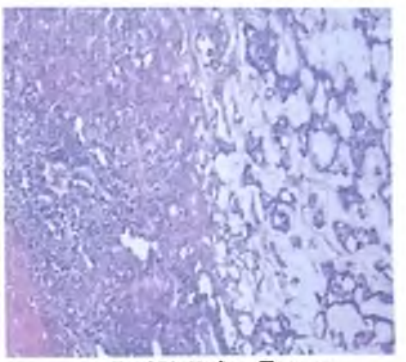
Incidence: most common testicular tumor of infants and children

MAC: non encapsulated and with homogeneous, yellow-white, mucinous appearance.

MIC: Schiller-Duval bodies and histochemistry can detect AFP.

Spread: early lymphatic

Special character: secrete alpha fetoprotein .
Good prognosis in children



MIC of Yolk Sac Tumor

II- Non germ cell tumours:

A- Sex cord stromal tumors: (mostly benign)

1- Prepubertal tumours:

Origin: from Leydig's cells.

Incidence: It usually occurs before puberty.

Special character: It secrets excess amount of androgen that results in precocious puberty with premature closure of the epiphyses and muscle hypertrophy, the so called *infant Hercules.*

2- Post pubertal tumour.

Origin: from Sertoli cells:

Incidence: It usually occurs after puberty.

Special character: It secrets oestrogen that causes gynaecomastia, loss of libido & feminization

B- Lymphoma:

Origin: from the lymphoid tissue of the testis.

Incidence: it occurs in elderly.

Special character: it tends to be bilateral and tend to invade the blood vessels.

***Spread of testicular tumour:**

1- Direct spread: *Early*: to the rest of the testis, epididymis, tunica & skin of the scrotum.
Late: to the spermatic cord.

II- Lymphatic spread:

- Cancer spreads directly to the para-aortic lymph nodes → cisterna chili →thoracic duct & may reach Virchow's gland.
- If the epididymis is involved, malignant cells may reach the internal iliac LNs via the lymphatics of the vas.
- If the scrotum is infiltrated (late), malignant cells may reach the inguinal LNs.

III- Blood spread:

-It is early especially in teratoma and choriocarcinoma.

-It is commonly to the lung " Canon ball metastasis " especially in choriocarcinoma, also to the liver, bone & brain.

***Staging:**

Stage I: - Localized tumour in the testis with no evidence of spread.

Stage II: Involved lymph nodes below the diaphragm.

Stage III: Involved lymph nodes above the diaphragm.

Stage IV: Distant metastasis.

Clinical picture:

History:

History of trauma in 10% of cases. Trauma only attracts the patient's attention to the mass.

Symptoms:

1- Painless, and rapidly growing scrotal (testicular) swelling.

2- Pain (usually late) in 30% of cases. It is dragging pain.

3- Symptoms of metastasis e.g. haemoptysis & pathological fracture may be the first presentation.

Signs:

General exam:

- Signs of distant metastasis e.g. *Troiser's sign.*
- Palpable enlarged abdominal LNs (above the umbilicus).
- Gynaecomastia in feminizing tumours and short stature in Leydig's cell tumour.

Local exam:

-The testis is enlarged; heavy with variable size & consistency with early loss of testicular sensatior

- The epididymis: early, it is normal but later, it is infiltrated.
- The vas is always free at the neck of the scrotum.
- Secondary hydrocele is present in 10% of cases detected by pinching test.

Investigations:

I- Tumour markers:

1) Human chorionic gonadotrophins HCG in urine (similar to pregnancy test done for fem

- The test is positive especially in choriocarcinoma (Normally up to 100 IU).

2) Alpha fetoprotein in the serum.

-By radioimmunoassay. It is positive especially in embryonal carcinoma.

3) Lactate dehydrogenase LH. enz.

These tumour markers are useful in follow up during & after treatment

II- Scrotal U/S.

III- Biopsy:

1- Needle biopsy is contraindicated as it may cause implantation of malignant cells to the scrotum and hence, spread to inguinal LNs.

2- Open (incisional or excisional) biopsy:

- Through *inguinal* incision, the spermatic cord is clamped with a vascular clamp and the tes is delivered through the wound and split and any suspicious mass is biopsied for frozen section examination. If malignant, orchidectomy is done in the same sitting.

IV- Investigations to detect metastasis:

1- Abdominal U/S, MRI & CT to detect LNs enlargement & hepatic metastasis.

2- Chest X ray.

3- Bone scan and lymphography.

4- IVU: -To detect enlarged LNs altering the course of the ureter or compressing the renal pelvis.
-To localize the kidney and mark its position in order to shield it durin radiotherapy.

Different diagnosis:

From other intrascrotal swellings (see later) especially $ orchitis as it also presents with enlarged testis and loss of testicular sensation. The testis is light (not heavy as in testicular tumours).

Treatment:

I- Retrograde high orchidectomy with block dissection of the para aortic LNs.

- Through inguinoscrotal incision.
- The cord is ligated and divided at the level of the deep ring and the testis & the cord are removed.
- Block dissection of the retroperitoneal LNs. Improves the prognosis in early cases but, it may cause failure of ejaculation due to removal of the lumbar sympathetic ganglia.

II- Orchidectomy + Postoperative radiotherapy (the treatment of choice).

- According to the type and the stage of the tumour.

A- Seminoma (highly radiosensitive):

Stage I: Radiotherapy to the para aortic LNs by inverted Y irradiation.

Stage II: Extend the radiation to the mediastinal & supraclavicular LNs.

Stage III & IV: Chemotherapy with or without radiotherapy.

B- Teratoma: (Less radiosensitive)

Stage I: only follow up by marker and abdominal MRI.

Stage II, III & IV: Combination chemotherapy including Cisplatinum, Methotrexate & Vincristin. Others prefer to add bleomycin.

Prognosis:

The 5-year survival rate is:

	Stage I & II	**Stage III & IV**
Seminoma	95 %	75 %
Teratoma	80 %	60 %

Differential diagnosis of the intrascrotal swellings.

I-Cystic swellings.

A- Translucent:

1- Hydrocele (the commonest).

2- Encysted hydrocele of the cord.

3- epididymal cyst

B- Opaque:

1- Haematocele.

2- Pyocele.

3- Chyocele.

4- Spermatocele.

Item	Hydrocele	Haematocele	Pyocele	Chyocele
History of trauma	May be +ve	+ve	May be +ve	-ve
Fever	-ve	Mild or absent	Marked	Absent or elephantoid
Tenderness	Not tender	Tender	Very tender	Not tender
Translucency	Translucent	opaque	opaque	opaque

II- Solid swellings.

A- Testicular swellings.

1- Syphilitic orchitis.

2- Testicular tumours.

B- Cord swellings.

1- TB. epididymis. 2- Bilharizial epididymitis .

3- Flarial nodule of the cord.

	TB. mass	B. mass	Filarial mass
History	-TB. toxemia -Symptoms of urinary TB	-History of contact with infected water. - Urinary & intestinal B .	-Filarial district. -Elephantoid fever . -Lymphoedema of the LL.
Site	The epididymis	Epididymis & the lower cord.	Any part of the cord.
The mass	Beaded or irregular	Pyramidal or oval.	Tubular & diffuse.
The cord	not amalgamated	Not amalgamated.	Amalgamated.
The vas	Beaded & thickened	Free (felt).	Not felt.
Tunica	Hydrocele is common	Hydrocele (rare).	Hydrocele or chylocele.
Scrotum	Atrophied and may show sinuses	Normal.	Usually shows lymph-oedema.

Chapter XI

Surgical ethics & Surgical audit

Surgical ethics

Ethics and surgical intervention must go hand in hand. The surgeon's intention is to cure or manage illness, and any bodily invasion that it incurs is only with the permission of the patient.

principles of medical ethics:

a. **Beneficence**: The intent of doing good and offering net benefit by therapeutic intervention.This principle requires maintenance of professional knowledge and skill by ongoing educationthroughout one's career.

b. **Nonmaleficence**: The intent of not causing harm. To exercise this principle, one must know probabilityof harm from a proposed intervention.

c. **Autonomy**: Deliberated self-rule requires a physician to obtain informed consent, respect privacyof healthcare information, communicate in a forthright manner that promotes trust, and respectpatients' wishes regardless of their alignment with the physician's own beliefs.

d. **Justice**: Acknowledgment of the patient within the context of society. This principle strives tocreate equitable treatments for similar patients and distribute resources as evenly as possible amongst those in need

Informed consent

*Autonomy of the patient mandates giving consent from patient to his surgeon before any surgical procedure. Information must be given clearly and confidentially before consent.

* A doctor performing a procedure on a patient without their consentcan be found guilty of battery (اذى بدنى).

*A doctor who has failed to give the patient adequate information toallow them to give informed consent can be found guilty of negligence (همال!).

*Essentials for informed consent:

- Confidentiality
- a quiet venue for discussion should be found;
- Respect patient'slanguage. avoid overly technical language in descriptions and explanations;

- Done by the surgeon or his deputy
- No stress or duress اكراه
- Sufficient amount of information must be given to mentally competent patient

* Five aspects the patient must understand to give informed consent:

- **The reason for carrying out the procedure**: The patient needs to understand the nature of their illness and its prognosis.
- **What the procedure involves**: Where and how long is the scar; what is being removed; what prosthesis will be implanted; will there be drains?
- **The risks of the procedure**: Specific to the procedure (e.g. stoma, limb dysfunction) and in general (e.g. anaesthesia, bed rest, deep vein thrombosis (DVT)).
- **The benefits of the procedure**: Improvement in symptoms or prognosis or purely diagnostic.
- **Alternatives**: Including conservative treatment, with their advantages and disadvantages.

Confidentiality

*the principle is that the information the patient reveals to surgical team is private and has limits on how and when disclosed to a third party.

*surgeons must not discuss clinical matters with relatives, friends, employers and others unless the patient explicitly agrees

*Confidentiality can be broken in special situations e.g. if it poses a serious threat to the health and safety of others.

Surgical audit(التدقيق)

" from Greek oudire ... to hear"

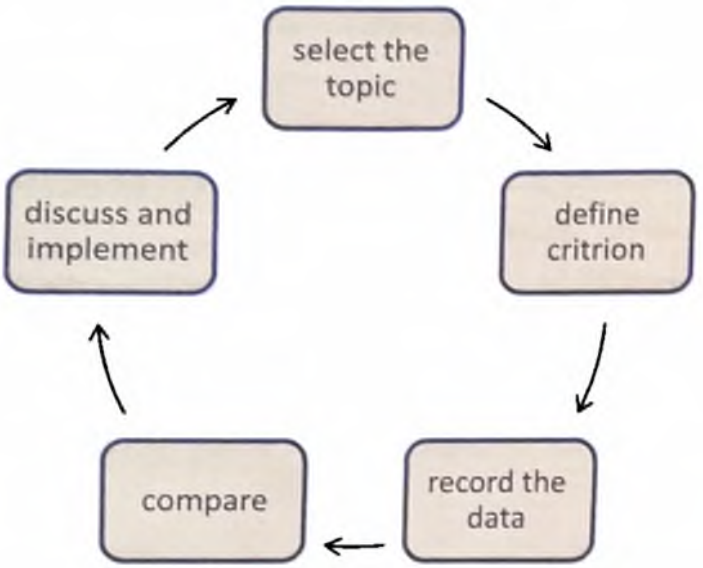
Definitions:

- Clinical audit is a process that aims to improve the care of patients and their outcomes by systematically reviewing the care that they receive and comparing this to certain defined criteria that may result in changes being made.
- Surgical audit: refers to the process of systematicallyexamining the procedure concerned and comparing it withthe 'gold standard'.
- Surgicalaudit provides the opportunityto highlight and address problem areas within the surgicalcare system. Through the implementation of any necessarychanges, surgical care can be improved
- Audit cycle:
  - it is a five steps scenario for analysis of a problem or aprocedure.
  - The last step of the cycle can guide a new cycle for an emerging concealed problem
  - If the first or second audit cycle revealed a problem or deviationfrom gold standards, a change of the procedure or new guidelinesare implemented
  - a new audit cycle then starts to confirm the benefit of the new guidelines
  - if the new guidelines are established, further cycles can be done to confirm adherence to standards and to discover other problems

Audit cycle

1) Select the topic for the audit : identification of a problem or area of concern .e.g. post appendicectomy wound infection
2) Clearly define the criterion and the standard: the problem must be clear for all e.g wound infection must be confirmed by clear data such as leucocytic count, wound swab etc. Also, the standard must clearly describe the accepted evidence based levels of care e.g. accepted rate of wound infection.
3) Record the data: clear and accurate data collection from patient'sfiles, investigations, operative notes and post operative charts.
4) Compare the audit results against the criterion and the standard: this could facilitate identification of the problem or open the way to make a new audit cycle about suspicious cause of the problem.

5) Discuss results and implement a change or a new cycle



Audit versus research:

Research asks: Are we singing the right song?

Audit asks: Are we singing this song right?

research	audit
Creates new knowledge	Test care given against knowledge gained from research
Based on hypothesis	Measure against standards
Results are generalizable and puplishable	Results are only relevant locally

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